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Selected Lectures and Essays, including the Fourth Edition of Ligaments, their Nature and Morphology.

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THE first part of this work contains a new edition of "Ligaments," their nature and morphology. The edition has been carefully revised, and in parts re-written. There are three new chapters—slings and pulleys, legs, wings and flippers, and the muscles of the orbit. Nearly all the blocks of the original (1887) edition are replaced by new. In re-publishing "Ligaments" the author yields to repeated requests from anatomical teachers of experience.

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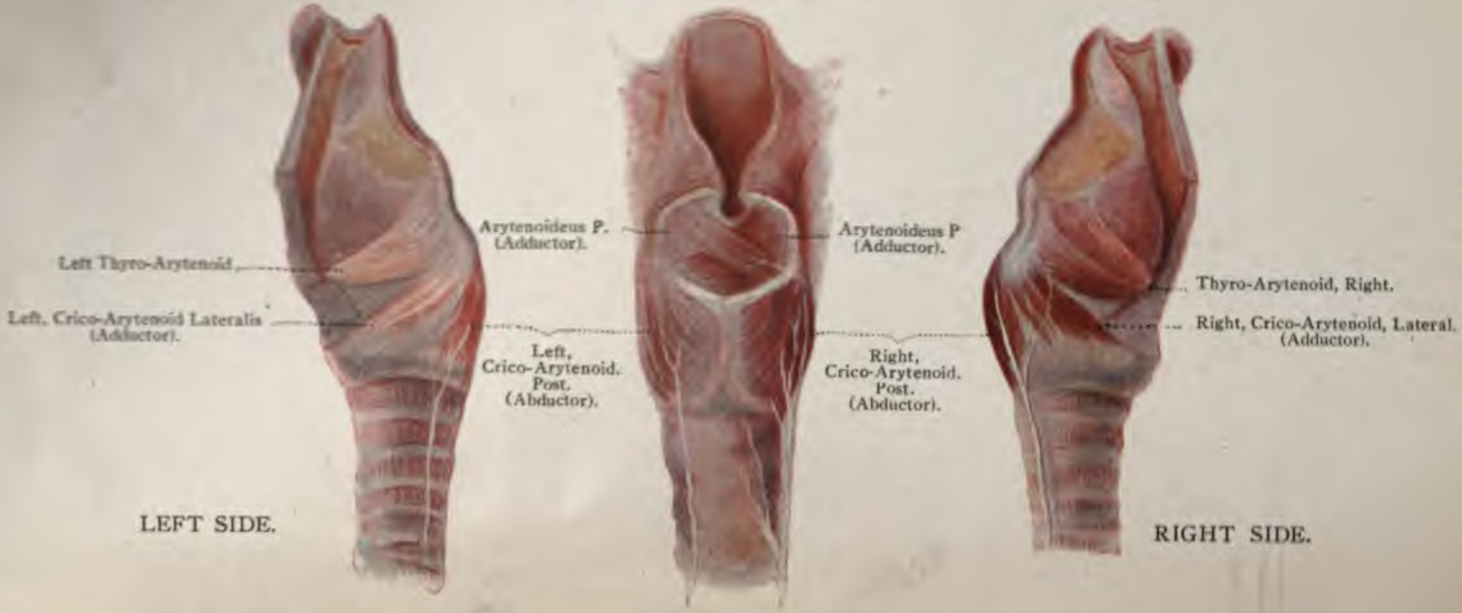
ANEURYSM OF AORTA WITH

Pressure on Left Recurrent N.


Left Thyro-Arytenoid
Left, Crico-Arytenoid Lateralis (Adductor).
Arytenoideus P. (Adductor).
Left, Crico-Arytenoid. Post. (Abductor).
Arytenoideus P (Adductor).
Right, Crico-Arytenoid. Post. (Abductor).
Thyro-Arytenoid, Right.
Right, Crico-Arytenoid, Lateral. (Adductor).
LEFT SIDE.
RIGHT SIDE.

Paralysis of left recurrent laryngeal nerve with paralysis and atrophy of the left side of the larynx. Above is the laryngoscopic image during quiet respiration (Fig. 1) and on attempted phonation (Fig. 2). From a case of the late Sir Morell Mackenzie's, painted by the late Lennox Browne, vide Trans. Path. Soc., Lond., 1870, xxi. p. 129.

I am indebted to Mr. E. Mayer for permission to use this drawing.

DISEASES
OF THE
THROAT, NOSE AND EAR

BY

DAN McKENZIE, M.D., F.R.C.S.E.,

Surgeon, Central London Throat and Ear Hospital; Oto-Laryngologist to The French Hospital, London; Hon. Cons. Laryngologist to the Emergency Hospital, Ilford; Editor of "The Journal of Laryngology, Rhinology and Otology."

WITH TWO COLOURED PLATES AND 199 FIGURES IN THE TEXT.

ST. LOUIS
C. V. MOSBY COMPANY
1921

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1921

PREFACE

THIS book has been written from the practical point of view, and for that reason many interesting scientific problems and issues have necessarily been omitted. On the other hand, considerable attention has been devoted to operative surgery, but here, also, limitation of space has rendered it impossible to describe the many modifications and variations of the operations.

In addition to the acknowledgments for the use of illustrations borrowed from other sources, I desire to offer my sincere thanks to Dr. Robert Knox, Dr. Andrew Wylie, Dr. William Hill, Dr. Irwin Moore, Mr. W. M. Mollison, and Mr. J. Gay French for kindly granting me the loan of valuable blocks.

The original illustrations are for the most part the production of Mr. S. A. Sewell, who has succeeded admirably in giving pictorial expression to my suggestions and ideas.

For the photographs, I am indebted to the careful skill of Mr. J. Peach, of the Central London Throat and Ear Hospital.

The original of the coloured frontispiece is one of the drawings executed for Sir Morell Mackenzie by Lennox Browne, for long in the possession of Mr. E. Mayer, of Messrs. Mayer and Phelps, and recently presented by him, together with manuscripts and instruments belonging to Mackenzie, to the Royal Society of Medicine. As far as I am aware, the drawing has never before been reproduced.

I am deeply conscious of the debt I owe to Mr. Mayer for his generous permission to use this historical picture, and I am confident that all readers who benefit by its clear and graphic representation of the laryngeal features in recurrent laryngeal paralysis, will share my feeling of gratitude.

51335

PREFACE

The case to which the drawing refers, by the way, is recorded, as Dr. Irwin Moore has discovered, in the Transactions of the Pathological Society of London in 1870 (vol. xxi., p. 129). The following is the title and some of the notes:—

> *Aneurism of the arch of the aorta, causing pressure on the left recurrent laryngeal nerve, with paralysis and atrophy of the muscles of the left side of the larynx.*
>
> Specimen shown at the Path. Soc. of London by Morell Mackenzie. April 19th, 1870.
>
> Laryngoscopic examination showed the left vocal cord immovably fixed between the side of the larynx and the median line and neither abducted in inspiration (Fig. 1), nor adducted on attempted phonation (Fig. 2).
>
> P.M.—The muscles on the left side of the larynx were markedly atrophied. The left crico-arytenoideus posticus, was considerably paler and thinner than its fellow. The fibres on the left side of the arytenoideus proprius were also less developed than those on the other side, but the greatest difference was observable in the crico-arytenoideus lateralis, the left muscle being nothing more than a few pale, scattered fibres, whilst the right muscle was large and fleshy. This difference in the condition of the several muscles is quite in accordance with the clinical history, as the voice was affected long before the breathing became impeded."

Finally, I desire to express my thanks to Dr. Wyatt Wingrave and to Mr. Archer Ryland for their kind help in the arduous task of proof correcting.

D. M.

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DISEASES OF THE THROAT, NOSE AND EAR

CHAPTER I

NOTE-TAKING AND GENERAL SEMEIOLOGY

Instruments.—No attempt will be made to give a detailed account of the many instruments used in the diagnosis of disease of these special regions, only those being mentioned in the text which the author himself has found most useful; and that for two reasons; first, because the simpler the student's armamentarium is, the less is he likely to be confused and troubled with a multitude of unnecessary details; and secondly, because each workman must gather together his own bag of tools for himself.

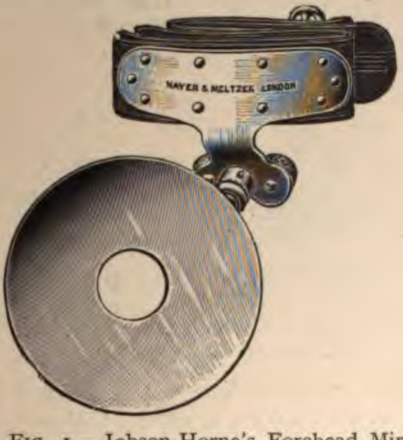

FIG. 1.—Jobson-Horne's Forehead Mirror.

Mirror.—Everyone has his own favourite mirror. The writer prefers one with a large eye-hole (Jobson-Horne's) as being infinitely the most comfortable to use for any length of time (Fig. 1). The focal distance should not exceed seven inches.

Lamp.—No one need hesitate to examine a throat or an ear for want of a special lamp, although it must be admitted that for operating in these regions, as well as for examination of the less accessible areas, specially powerful illumination is a desideratum, and indeed a necessity. Of these lamps, the Nernst is perhaps the most powerful, and, before the outbreak of the Great War, it was the light most commonly used in spite of its cost and fragility. A substitute, known as the " Half-Watt," is now being made in England, and I can recommend it as being almost as strong as the Nernst, and much more durable.

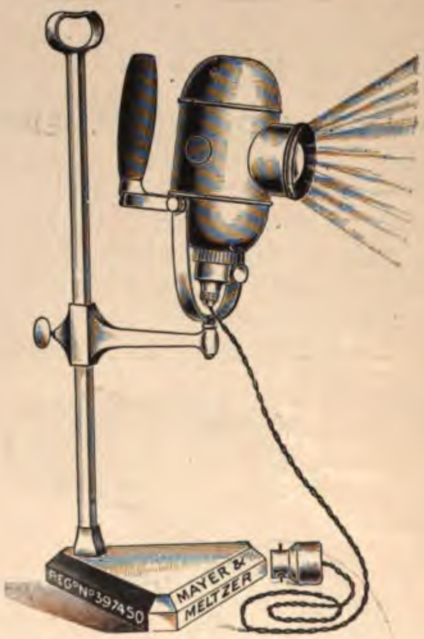

FIG. 2.—Mayer & Phelps' Examination Lamp.

As a travelling lamp, one uses the electric headlight, supplied from an accumulator or from the main.

In an emergency, an ordinary paraffin or incandescent gas light serves the purpose very well, and direct sunlight (when it is obtainable) supplies us with brilliant illumination. Care should be taken when using sunlight to avoid focussing the rays too intensely upon the spot under examination, or a burn may be produced.

Practitioners in remote districts will find the small paraffin lamp with a parabolic reflector, as advertised in the medical journals, of considerable use, provided that the reflector does really reflect.

Small electric torches are useful for directly examining the pharynx, and the more powerful varieties may be used to supply illumination for a head-mirror.

The light for the mirror should be placed on a level with and just behind the patient's left ear, so that it shines full on the examiner's face as he sits opposite to his patient. If the examiner is unprovided with a mirror, the light should be held by an assistant, just on a level with the former's right ear, and with the rays directed full on the patient's face.

The methods of illumination used in the various forms of endoscope will be dealt with later.

Considerable practice is required before one can manage the head-mirror and light with freedom and success, and this is specially true of operating. For that reason, the practitioner and student should attend the special hospitals and clinics as clinical assistant in order to become adept in the use of the mirror and head-light, an accomplishment which, it should be noted, is of the utmost value, not only to the specialist, but also to the general practitioner, and in the examination and treatment of regions other than the throat, nose and ear.

Mask.—It is advisable always to wear a light mask of sterilized material when examining patients.

Note-taking.—A few remarks upon note-taking as practised at the Central London Throat and Ear Hospital will, first of all, be made.

Following the headings in the out-patient's letter, we find the first space to be filled up is that dealing with the patient's

Complaint.—What is the most prominent subjective symptom ? Endeavour to obtain from the patient a definite statement as to what ails him. Do not be satisfied with vague answers, like " Something in my nose," " My throat is bad," and the like. Do not accept ready-made diagnoses, such as " ulcerated throat," " polypus of the nose," etc., but ascertain what the patient actually feels amiss. It should be remembered that certain terms mean one thing to the doctor, and something else to the laity. Thus, when people say they have a " discharge from the ears," they may be referring to small pieces of cerumen which occasionally find their way out of the canal after washing. If a man is suffering from aural discharge, he will say he has a " running ear." In like manner, the examiner will often be surprised to find patients deny that they are " deaf," but quite ready to admit that they are " hard of hearing." In the case of aural discharge, be careful to discriminate between discharges which are watery (" thin," " colourless," " white ") and those which are purulent (" thick," " yellow," or " mattery ").

Ascertain whether the *onset* of the illness has been *sudden* or *gradual*.

Certain diseases are characterized by a sudden onset: thus, sudden loss of voice, when occurring in a female patient, will often be found to be due to hysterical paralysis of the adductors of the cords, and the sudden occurrence of marked deafness, without pain, suggests cerumen as likely to be the cause. Acute or subacute disturbances in the nose, pharynx, larynx, ear, etc., are generally sudden in their onset; and, on the other hand, obstructive deafness from chronic catarrh of the middle ear, or from what is known as " otosclerosis," is of insidious and gradual onset. Occasionally, however, the onset may appear sudden to a patient of moderate intelligence, inasmuch as a considerable degree of deafness in one ear may exist without his being aware of it, and it is only after the function of the unaffected ear has become impaired that the patient discovers his infirmity. Of gradual onset, we find hypertrophic rhinitis and chronic complaints generally. The hoarseness of simple chronic laryngitis and of tuberculous and syphilitic laryngitis is gradual in its development, the voice losing its finer qualities, and becoming rough and harsh in tone before it is finally lost.

It is, of course, of the greatest importance to ascertain whether an acute condition has supervened upon a chronic ailment, such as the occurrence of a recent attack of severe earache in a case of long-standing running ear, and it very frequently happens that this vital fact is only discovered by close questioning. (See p. 475.)

Duration of Complaint.—A definite statement should, if possible, be obtained, and for this patience and some skill in cross-examination are required. Expressions like " some time," " some months," are naturally valueless. It is important to find out whether the ailment is continuous or intermittent (" on and off "). In intermittent complaints—e.g., in intermitting suppuration of the middle ear—patients always give the duration of the present attack only, and unless cross-examined, will lead the student astray, stating as duration of the illness weeks, when it should be years.

Voice.—For slight alterations in the tone or quality of the voice, one must accept the patient's word. In ordinary cases, dealing with people who are not in the habit of observing their bodily functions closely, considerable departure from the normal may exist without their being aware of it; but in the case of singers, actors, public speakers, etc., the observation of the patient regarding alterations in the *timbre* of his voice may be of considerable value. Ascertain which "register" is most affected, and whether the roughness or weakness affects the chest notes or the head voice. The examiner can observe for himself many abnormalities of the voice. Note, first, whether the voice is well produced, whether the sound is thrown well forward *ore rotundo*, or whether the speaker phonates with mouth half closed, or in an affected manner, throwing the voice up, as it were, into the naso-pharynx.

In addition to these errors in voice-production, accustom yourself to the definitely abnormal voice—e.g., a toneless, dead character in the speaking voice betokens obstruction in the naso-pharynx or nasal cavities. This is frequently miscalled the "nasal voice." The true nasal voice (speaking through the nose) is heard in quite contrary conditions—in cleft or perforated palate, in enlargements and tumours of the tonsils and fauces, and in paralysis of the soft palate, in all of which the vocal vibrations are directed into the nasal cavity instead of through the mouth. Note, further, any interference with pronunciation—with labials, in paralysis affecting the orbicularis oris muscle; with dentals and linguals, as in the mumbling speech present when the tongue is tied down by adhesions or new growths to the floor of the mouth; with gutturals, like "ing," in nasal obstructions, etc. The presence of adenoids seems to predispose to abnormalities in the speech of children, and there is a particular irregularity known as the *nasal lisp*, in which the "s" sound is produced by the sides of the tongue or in the pharynx, and which is said to be always associated with them.

In addition to these commoner changes in the sound of the speaking voice, there is another much more rare, but so characteristic as to be unmistakable. It consists in the acquisition by the voice of a curious hollow tone—hence we may call it the *cavernous* voice—from the presence of some considerable obstruction at or about the base of the tongue, the epiglottis, the posterior pharyngeal wall, or elsewhere where it presents an obstacle to the emission of sound about the vestibule of the larynx.

The tones of the voice in patients who are very deaf are frequently flat in quality, and syllables and words are clipped in such a manner as to impart to the speech a curious staccato sound. This is probably due to the patient's inability to hear his own voice. The tones by means of which we impart depth and character to the voice, not being heard or appreciated by a deaf man, are not sought for by him to give to his speaking voice the colour proper to his mental attitude.

Hoarseness is a symptom of great importance, and should always lead to an examination of the larynx with the laryngoscope. There is no disease of the larynx from simple catarrh to cancer which may not cause hoarseness. But the converse is also true; there may be serious laryngeal disease without any change whatever in the voice. The reason for this is, of course, that hoarseness is due to impairment of the action of the vocal cords, and is only present when these structures or the muscles moving them are involved in disease.

The commoner varieties of hoarseness are, the coarse, rough voice of chronic laryngitis and pachydermia laryngis, varying in degree from the mere " port wine " voice up to one of a highly raucous quality; and the feeble voice of laryngeal tuberculosis, which, in the later stages, loses all trace of phonation, and becomes a mere harsh whisper. When all phonation has disappeared, we speak of the condition as *aphonia*. As already indicated, hoarseness (dysphonia) tends to result in complete aphonia. But aphonia may appear without any precursory hoarseness in functional or hysterical paralysis of the vocal cords, and also in simple acute catarrh.

Recurrent hoarseness in children is usually catarrhal and secondary to adenoids, but continued hoarseness in early life should raise suspicion of papilloma of the larynx.

In adults over middle age, continued hoarseness should lead to an examination of the larynx for cancer.

Cough.—The dependence of cough upon abnormal conditions of the upper respiratory tract should always be remembered. Note its frequency, and whether or not it is accompanied by expectoration. Ask the patient to cough, and observe its character, whether " rough," " croupy," or " brassy," as in laryngeal affections. Inquire into the nature of the expectoration—whether mucous, muco-purulent, purulent, blood-stained, etc. When a patient complains of " bringing up" blood or phlegm, ascertain whether the morbid material is "coughed up," "hawked up," or "thrown up." As a matter of fact, it is nearly always difficult and sometimes impossible to be sure, when a patient complains of " spitting blood," where it is coming from. Most frequently it is genuine hæmoptysis, and is proceeding from the thoracic organs, but true hæmoptysis may be very closely simulated by blood from the upper air passages finding its way into the larynx and trachea. Thus it is met with after epistaxis and in cases of adenoids, as these growths are friable and bleed easily. Occasionally the blood comes from the mouth, either from the gums, especially in pyorrhœa, or from a dilated capillary at the base of the tongue. In such cases a complaint that the blood comes into the mouth after eating, or after sucking sweets, may lead us to a correct diagnosis.

In all cases, however, the complaint of blood-spitting should lead to examination of the chest, even although bleeding spots in the mouth or upper air passages have been found.

Almost any disorder of the throat, nose or ear may induce cough, and the cough arising therefrom is usually " dry "—i.e., unattended by expectoration. Of this nature is the cough due to the irritation produced by cerumen or foreign bodies in the external auditory meatus ; the cough due to elongated uvula, which is usually most troublesome when the patient is recumbent ; the cough arising from such intranasal conditions as polypi, etc. This cough is often described as " hacking," and is one variety of the old-fashioned " stomach cough." Cough set up by these conditions is usually paroxysmal, and may be very severe ; so severe, indeed, are the paroxysms at times that they culminate in vomiting, and so may simulate whooping cough. Not seldom the cough is the sole symptom for which the patient seeks relief, and it is then our task to discover, if we can, the source and origin of the attacks. In addition to the causes already mentioned, search should be made for " cough-spots " in the nose. They occur at the " tubercle " of the septum, and at the posterior and anterior ends of the inferior turbinal body (in order of frequency). Another common site of origin lies in the " trigger area " of swallowing at the base of the tongue. (See also later, p. 21.)

In hysterical aphonia the complete absence of sound during efforts at speaking is often associated with a perfect cough, in which the observer may detect phonation.

Respiration.—The patient should be asked to breathe through the nose. As a method of ascertaining the degree of patency of the nasal cavities, Wingrave's Spirograph test is both simple and satisfactory. It consists in making the patient breathe out through the nose upon a polished surface, such as a thin slab of polished wood or vulcanite, preferably of a dark colour, held horizontally just below the nostrils. The condensation of moisture forms a cloud on the polished surface, and as this evaporates, the portion corresponding to the obstructed nostril disappears before that corresponding to the open side. By a constant use of this test, it is possible to form a rough standard by which one can determine the degree of obstruction present in one or the other or both sides.

Nasal Obstruction is a frequent complaint. Ascertain whether it is permanent or variable. The former is found with nasal polypi, new growths, deviations of the septum, adenoids, and such diseases or injuries as induce permanent swelling of the nasal mucosa.

Variable obstruction is generally due to turbinal engorgement, which typically comes and goes at irregular intervals. As a rule, a change in the temperature of the air, whether from hot to cold or *vice versâ*, is sufficient to bring about a change in the nose, either for better or for worse. Patients often complain of *alternate obstruction*, one side being patent while the other is closed. This is particularly remarked on lying down, the blocked side corresponding with the side the patient is lying on.

Nasal obstruction gives rise to many serious secondary effects, such as Eustachian obstruction, catarrh of the middle ear, chronic pharyngitis and laryngitis, and even chronic bronchitis and asthma.

Such conditions are most frequently seen when the nasal obstruction is itself secondary to serious local infections, such as sinus suppuration, but the milder forms of nasal and sinus catarrh are also apt to induce them. It is generally believed that even the milder forms of nasal catarrh are due to microbic infection, and that these aural, laryngeal and bronchial phenomena are due to extension of infection. It must not be forgotten, however, that simple anatomical abnormalities, such as deflections of the nasal system, may be the starting point of persistent changes, proximate as well as remote, of an infective character.

Ascertain if the patient is a *mouth-breather*. Does he snore when asleep? **Snoring should be always regarded as a symptom of disease.** In children, snoring generally means adenoids with or without enlarged tonsils; in adults, the symptom is usually due to elongation and engorgement of the uvula and paresis of the soft palate. People with marked nasal obstruction, also, generally snore when asleep. Frequently, no information can be obtained regarding a patient's breathing during sleep, but the complaint of "dry throat and mouth in the morning" may be looked upon as an indication that the patient is a nocturnal mouth-breather. Healthy, normally constituted people are unable to sleep while breathing solely through the mouth, but the ability to do so is easily acquired, especially when the obstruction to the normal passage of air through the nostrils is of slow development. New-born infants always breathe through the nose during quiet respiration, and never through the mouth, unless when crying. As a consequence, nasal obstruction of sudden onset at this period may directly menace life, not only from the interference with suckling, but also from the circumstance that the baby begins to cry, and keeps on crying as long as it breathes through the mouth, so that rest and sleep are unattainable, and the child, if unrelieved, may die of exhaustion.

Stridor.—Interference with the current of air, from disease or a foreign body, as it passes through the larynx or trachea may set up stridor, an unmistakable sound to those who have once heard it. When produced in the larynx, stridor accompanies inspiration; when proceeding from the trachea, it accompanies both inspiration and expiration. In the latter case we may suspect the cause to be aneurysm, goitre, or tertiary syphilis affecting the tracheal wall. The obstruction to the breathing may be so great as to cause dyspnœa on exertion, during which the stridor becomes louder, as it does in a "roaring" horse. Remember the occasional occurrence of *congenital stridor* in new-born infants. (See p. 96.)

Diseases of the nose and naso-pharynx are frequently associated with paroxysmal asthma and bronchitis.

Attacks of Dyspnœa with Stridor.—Choking sensations or attacks arise from a multiplicity of causes. They vary in severity, from a mere transient feeling of suffocation to actual asphyxia, ending in unconsciousness, and perhaps death. The causes giving rise to this symptom may be tabulated as follows :—

1. *Slight choking sensations*, usually reflex, occur in—
   - (a) Enlargement of the lingual tonsil.
   - (b) Paræsthesia of the pharynx or larynx (globus hystericus).
   - (c) Anæsthesia of pharynx or larynx.
   - (d) Goitre, simple and exophthalmic.

2. *Severe attacks of asphyxia* from physical obstruction to the passage of air through the pharynx, larynx or trachea may arise from—
   - (a) Foreign bodies in the pharynx, larynx or trachea.
   - (b) Membranous laryngitis.
   - (c) Simple, tubercular, or syphilitic laryngitis.
   - (d) Pharyngeal or laryngeal neoplasms.
   - (e) Bilateral paralysis of the abductors of the vocal cords from pressure on both recurrent laryngeal nerves, by tumours, goitre, aneurysm, etc.
   - (f) Spasm of glottis.
   - (g) Direct pressure on larynx, trachea or large bronchi, by tumours, goitre, aneurysms, etc.

Deglutition.—Ascertain in cases where difficulty or pain is experienced on swallowing, whether trouble arises on swallowing liquids or solids. It is a good plan to make the patient swallow in your presence, giving him water to drink or some food to eat, according to his complaint. *Dysphagia* may arise from pain, from paralysis, or from some mechanical interference with deglutition. A raw, scraping soreness on swallowing characterizes the several forms of pharyngitis, and in this case, as a rule, it is less painful to swallow food than saliva. Severe pain is a frequent and distressing accompaniment of tubercular ulceration of the larynx if the epiglottis or other parts about the entrance to the larynx have become involved in the disease. Interference with deglutition from inflammatory conditions reaches its maximum in peritonsillitis or quinsy, when swallowing may become quite impossible, so that the saliva dribbles from the half-open mouth. In this instance the dysphagia is due partly to pain and partly to physical interference with the action of the pharyngeal muscles by the intense and massive inflammatory swelling. In paralysis of the soft palate (usually diphtheritic) imperfect deglutition may lead to a regurgitation of fluids through the nose, and the same disability is experienced in perforations of the palate, congenital or acquired.

Inability on the part of young infants to fasten to the mother's nipple is generally due to nasal obstruction from adenoids; but in older children also difficulty in swallowing should lead to an examination for these growths.

Obstruction to swallowing, more or less complete, occurs in stenosis of the œsophagus, malignant or simple, and in these cases likewise a regurgitation of food may occur, usually a very short time after it has been swallowed. A similar occurrence takes place when there is a pharyngeal pouch. (See p. 197.)

Pain.—The occurrence of pain is to be noted. Mark its situation, its severity, whether it is increased or relieved by pressure over the painful area, and by what muscular movements it is evoked or intensified. The pain of sore throat should be discussed, whether it is constant or felt only on swallowing. Diphtheria affecting the throat is often painless, and the same is true of the tertiary syphilitic ulcerations. In peritonsillitis the pain is constant, violent and unilateral, shooting up towards the ear of the affected side. Normally, indeed, tonsillar sensation is referred to the Eustachian tube.

In nasal sinus affections—often when acute, but seldom when chronic—pain, sometimes very severe, is complained of in the frontal region, the root of the nose and orbit, or the cheek, according to the sinus affected, in typical cases. The situation of the pain in sinus inflammations is, however, by no means constant; thus, pain in the forehead or root of the nose is frequently due to disease of the maxillary antrum, and Abercrombie has reported a case of unilateral frontal sinusitis in which the pain was referred to the supra-orbital ridge of the healthy side. In chronic sinusitis, the site of the pain is less well defined than in acute sinusitis; pain, indeed, apart from headache is usually altogether absent. When it is present it is often made worse by blowing the nose, coughing, sneezing, lowering the head, and the like.

With or without definitely localized pain, sinus suppuration is a frequent cause of severe *headache*, at times associated with drowsiness, vertigo, and mental confusion, almost amounting to loss of consciousness. The headache of maxillary antrum and frontal sinus suppuration is usually referred to the forehead; that of sphenoidal sinus suppuration to an area on the skull half way between the external auditory meatus and the external occipital protuberance.

One must also remember that " neuralgia," ascribed to carious teeth or to neuritis of some branch of the trigeminal, may arise reflexly from the nose, either from sinus disease, or, what is perhaps more frequent, from the long continued pressure-irritation of some bony septal spur or enlarged turbinal.

Headache may be the first symptom in meningitis arising in the course of aural or nasal disease; it is usually associated with stiffness in the muscle of the nape of the neck.

Laryngeal disease is painless so long as it is confined within the larynx, but when once the boundaries are crossed, the pain set up may be very severe. This is particularly true of cancer and tubercle, whether the extension made is into the pharynx or into the cartilaginous framework whereby perichondritis is induced.

There is one exception to the general mildness of laryngeal sensation, and that occurs in the familiar laryngitis of an " acute cold," the cough of which is often very painful, the pain being referred to the middle of the sternum.

Freedom from pain also characterizes œsophageal affections so long as they are confined to the œsophagus.

Turning to the ear, we find that most of the acute affections are painful; furuncle of the external auditory meatus, acute catarrh, acute suppuration of the middle ear, and acute mastoiditis, are all marked by severe pain, in children at all events. But in adults it is remarkable that severe inflammation of the middle ear may excite no pain whatever, and all that the patient notices is some deafness followed by the onset of discharge.

The deep-seated boring pain known as "earache" is generally due to inflammatory diseases of the middle ear. Its occurrence in sufferers from chronic ear discharge is of great importance, inasmuch as it may foreshadow or indicate an extension of the disease to the mastoid cells or to the cranial cavity. In typical cases it is worse at night.

The chronic diseases of the middle ear are usually painless, although occasional twinges are sometimes complained of.

In general, it may be stated that pain in the ear is a danger-signal; but, on the other hand, it must be remembered that dangerous diseases of the ear may exist without causing any pain.

Tenderness on pressure over the mastoid is a sign of great importance as an indication of mastoid disease. (See later, p. 494.)

Pain in the neck should lead to careful examination of that part. Palpate the glands at the angle of the jaw, noting whether they are enlarged or tender. Pain and swelling, with œdema of the skin, in any part of the neck should arouse suspicions of abscess under the middle or deep layers of the deep cervical fascia.

Taste and **Smell** may conveniently be taken together, as disturbances of one are generally associated with disturbances of the other. These disturbances are classified thus: *hyperosmia*, excessive sensibility of the olfactory organ; *hyposmia*, diminished sense of smell; and *anosmia*, absence of the olfactory sense. In like manner, the term *hypergeusia*, *hypogeusia* and *ageusia* apply to corresponding disorders of the sense of taste. A deficiency in taste is generally combined with a deficiency of smell, and that in turn is most frequently caused by some form of nasal obstruction, the removal of which will restore both functions. In cases where no sign of nasal obstruction exists, we proceed to test the olfactory and gustatory senses chemically. In testing smell, avoid the use of pungent substances, like ammonia, which stimulate the nasal terminations of the fifth cranial nerve. Essential oils, such as cloves, or odoriferous substances like musk or camphor are suitable. In testing taste, direct the patient to put out the tongue and close the eyes. After drying the tongue, apply to its surface, on a glass rod, a bitter (solution of quinine), sweet (sugar or saccharine), acid (dilute acetic acid), and saline (solution of common salt).

The sense of flavour is olfactory.

(The discussion of *deafness* and other auditory phenomena will be found at p. 576.)

The **Physiognomy** or **Facies** of the patient may at once guide us to the seat of the disease. One should accustom oneself to the facies presented in the various diseases of the regions under consideration. There are, for instance, the open-mouth, vacant expression, buck teeth, and toneless voice of the child with adenoids; the strained, listening expression, inclination of the better ear, toneless and clipped syllables of chronic deafness; the dribbling mouth, stiff carriage of the head, facial expression of pain and anxiety, together with the nasal phonation and the difficulty in opening the mouth, seen in quinsy.

The facies of throat cancer in the advanced stages is also characteristic with its open mouth, haggard and pallid face, fœtid breath and obvious emaciation.

In adults one associates broadening of the bridge of the nose with ethmoidal disease and nasal polypi, and sinking of the bridge of the nose with tertiary syphilis of the septal bones, while in ozæna we frequently see a wide, flat nose with open nostrils. In this disease also, a profile view of the patient will usually show that there is a little dimple on the bridge of the nose, in the middle line, just below the lower edge of the nasal bones, probably due to thinning and weakening of the septal cartilage, as the same slight deformity is produced when too much of the cartilage has been removed in the operation of submucous resection of the nasal septum.

Persons who have been subjects of hereditary syphilis bear the records of their past history in the radiating scars at the angles of the mouth, in the notched incisor teeth, and, it may be, in interstitial keratitis.

Again, the association of chronic deafness in an adult, with scanty hair, a dry skin, a fresh-coloured complexion, and a general appearance of stoutness, brings myxœdema to the mind.

The protrusion of one auricle in a patient who has suffered from discharge from the ear would suggest the likelihood of a mastoid or temporal abscess. (Fig. 171.)

Before bringing a speculum near the nose or ear, carefully inspect the patient on one side of his head and on the other, full face and from behind. (The last enables us to judge most surely of deformities or displacements of the auricle.)

General Examination.—The diseases and abnormalities in the throat, nose, and ear have a close connection with the patient's general health and constitution. Not only do these diseases lead on to general ill-health, but they may be in themselves the result of some general disorder. We have already made mention of myxœdema as a cause of deafness, but we must also remember that nerve deafness may be one phenomenon of a general nervous disease, and that suppuration of the ear may induce brain disease. In like manner the close association of nasal abnormality or disease with complaints like asthma, neuralgia, headache, epiphora, optic neuritis and other diseases of the eye; of pharyngeal disorders with the infectious fevers, pneumonia, herpes, and diabetes; of laryngeal affections with pulmonary tuberculosis, thoracic aneurysm, and other chest diseases, should never be forgotten.

Consequently, it may be laid down as a clinical rule that an examination of the throat, nose, and ear, and adjoining regions is incomplete until a general survey of the patient has been made.

Colds.—The liability of sufferers from throat, nose, and ear diseases to " colds " should be investigated closely, for under this term, people include all manner of troubles connected with the ears and upper respiratory tract.

CHAPTER II

EXAMINATION OF THE BUCCAL CAVITY, FAUCES, AND PHARYNX

PHYSICAL EXAMINATION

The Lips, etc.—Note the shape of the mouth when the patient is quiet, and when he is smiling or talking—whether, e.g., it is drawn to one side, as in facial paralysis. Next observe the lips, noting whether while the patient is at rest they are closed or open. Note also their colour—red, cyanotic, or pale. Frequently, nasal and pharyngeal catarrhs are accompanied by dry, peeling, or cracked and sore lips. Note also the presence of skin eruptions—herpes, eczema, etc.—on the lips, and of scars at the angles of the mouth.

Having inspected the lips, ask the patient to open the mouth. Difficulty or pain in doing so is found in—

1. Acute peritonsillitis;
2. Acute external otitis (especially furuncle);
3. Tonic spasm, from (a) tetanus, (b) strychnine poisoning, (c) reflex spasm from unerupted wisdom teeth or from dental caries;
4. Synovitis or arthritis of the temporo-maxillary articulation;
5. Ankylosis of the temporo-mandibular articulation;
6. Adhesions—e.g., following cancrum oris; gunshot and other war injuries;
7. Cancer of tongue, tonsil, pharynx.
8. Parotitis—epidemic, septic, etc.
9. Otitic pterygoid abscess (rare).

The mouth open, inspect the inner surfaces of the lips, where mucous patches of the secondary stage of syphilis occasionally appear. On this surface are to be found in the pre-eruptive stage of measles the so-called Koplik's spots. These resemble minute sago grains on a red ground sprinkled over the mucous membrane, to which they impart a granular appearance. They are most easily demonstrated when the light, which must be daylight, falls on the surface at a slant.

Having inspected the inner aspect of both lips, together with gingivo-labial recesses, transfer the attention to the state of the teeth, artificial dentures being removed if present. Look for the stunted, peg-shaped central upper incisors, with the notch in the free edge, indicative of hereditary syphilis—the "Hutchinsonian teeth." Examine carefully, under the reflected illumination of your forehead mirror, for signs of dental caries. To ensure accuracy, a careful inspection by means of a small laryngeal mirror in the inner and posterior aspects of the teeth is advisable. Carious teeth and pyorrhœa alveolaris are frequently responsible for the septic conditions of the buccal and pharyngeal structure, and carious disease of the upper molars is often found in conjunction with suppuration in the maxillary antrum. (See p. 325.)

The **Gums** must also be inspected. In addition to such alterations in colour as lividity or pallor, the gums may exhibit changes due to general diseases like scurvy and purpura, mercurial salivation, lead-poisoning, etc. Make a note of pyorrhœa alveolaris if it exists. The mucous membrane of the gums and cheeks is generally reddened in catarrhal colds, and a circumscribed patch of erythema may arise from the irritation of a jagged tooth, or from the existence of alveolar abscess. In mumps, the opening of Stenson's duct, opposite the second upper molar, assumes the appearance of a red papilla pouting from the surface of the cheek. The white curdy flakes of thrush are frequent on the mucous surfaces of the cheek in infancy, and towards the close of exhausting diseases in adults.

Next direct your light upon the **tongue**. Note its size and movements. Can it be fully protruded? Hindrances to protrusion may be due to (1) abnormal shortness of the frenum ("tongue-tie"); (2) ulceration of the frenum; (3) (in the substance of the organ) gummatous infiltration; (4) cicatricial contraction; or (5) cancerous disease, etc. The tongue is protruded towards the paralysed side in hemiplegia. Everyone is familiar with the jerky protrusion and snappy withdrawal of the tongue in chorea. Fine muscular tremors, as in alcoholism and other toxic states, and in certain nervous diseases, such as exophthalmic goitre, etc., are plainly seen in the tongue. Note the condition of the dorsum of the tongue, with its three varieties of papillæ—the fungiform, as tiny red points; the filiform, most numerous towards the tip; and the eight or ten circumvallate papillæ, arranged like the letter V diverging from the foramen cæcum, towards the back of the organ. These last are occasionally looked upon by patients as evidences of disease or as growths. Notice whether the dorsum is *furred*, and, if so, whether the furring is general or limited to one side or part. Localized or unilateral furring is usually due to carious teeth. One ought to be able to recognize the " strawberry " and the later " raspberry " tongue of scarlet fever; the large, pale, tooth-indented tongue of chronic dyspepsia and alcoholism; the " irritable " tongue, with its red islet on the dorsum from excessive tobacco-smoking; the " fissured " tongue; the " geographical " tongue; the " tertiary " tongue, with its areas of leukoplakia (see p. 603), etc. Valuable information may often be obtained from a careful investigation of scars on the tongue.

Examine next, in rotation (1) the hard palate, (2) the soft palate, passing on to (3) the uvula, (4) the anterior and posterior pillars of the fauces, (5) the tonsillar region, and (6) the posterior wall of the pharynx.

The Palate.—Making the patient tilt his head right back, note the shape of the vault of the palate—whether, e.g., it is highly arched and narrow, as in a neglected case of adenoids, in which case the teeth will probably be found to be irregular and prominent. In edentulous people after absorption of the alveolar processes, and in infants, the palate is flat. The palate may be perforated, cleft, or absent.

Notice next the movements of the soft palate and uvula. The first introduction of the tongue-depressor is, in most cases, sufficient to induce an instinctive elevation of the soft palate and uvula; but when paralysed, as in diphtheritic paralysis, the soft palate hangs motionless and irresponsive to stimuli, remaining stationary during deep inspiration, phonation, and deglutition. In unilateral paralysis the uvula is drawn towards the sound side.

Observe the colour of the palate, fauces, uvula, and pharynx. Pallor of these regions is observable in anæmic conditions, and in phthisis. If there is congestion, note its intensity and its distribution—whether general, or limited to certain regions, as, e.g., the tonsils. General hyperæmia is seen in alcoholism, excessive tobacco-smoking, gouty conditions and in simple catarrh. The congested pharynx of measles is associated with the appearance of the rash on the soft and hard palate as punctate areas of redness, with lines radiating from them in a stellate manner. In scarlet fever, the hyperæmia is intense, general, bright and uniform. The scarlet throat in severe cases looks glazed and dry, and is spotted with stringy flakes of mucus adhering to the posterior pharyngeal wall. A hyperæmia limited to the soft palate or to the pillars of the fauces and the tonsils, showing the so-called "snail-tracks," raises the question of secondary syphilis, but it may be added here that in certain cases with simple tonsil hypertrophy, an appearance is obtained very closely resembling the typical " snail-track " of the secondary throat.

Herpes is sometimes seen on the palate and pharynx. It should always be looked for in facial paralysis.

Uvula.—The uvula participates in all general inflammations of the pharynx. In very acute conditions it tends to become œdematous and translucent ; in long-continued inflammations it may undergo hypertrophy and elongation. The uvula may be bifid or congenitally absent. Absence may also have been brought about by tertiary syphilitic ulceration, and by the surgeon in the operation of enucleation of the tonsils. In the latter the uvula may be drawn up and absorbed, so to speak, into the body of the soft palate as a result of the scarring from an operation which has been perfectly well performed, the uvula itself being free from wounds and scars.

Examine the **Tonsillar Region**, depressing the arched tongue by means of a tongue-depressor. Fränkel's is the most useful all-round tongue-depressor (Fig. 3) ; but larger tongue-depressors are often necessary to control the involuntary movements of the organ, especially in nervous patients, and for children there is no tongue-depressor like the handle of a teaspoon.

Having warmed and gently introduced the sterilized tongue-depressor between the lips, ask the patient to phonate, saying " Ah-h-h," and while he is doing so, slip the depressor on to the dorsum of the tongue. A practical hint of importance is necessary here. Do not insert the tongue-depressor further than the foramen cæcum, otherwise a small sensory twig of the superior laryngeal nerve ramifying over the mucous membrane of the base of the tongue will be stimulated, and gagging and retching will ensue. Neither is it wise to be too timid, for if the depressor is placed on the tongue too near the point, a proper control of the organ will not be obtained. There is an exact spot just in front of the foramen cæcum on which the tip of the depressor should rest.

Having obtained a proper control of the tongue, direct your light first on one tonsil and then on the other. Note the size and shape of the tonsils. Do they seem larger than usual ?

In order to scrutinize each tonsil in turn, move the patient's head first to the left, and hold back the right corner of the mouth, and then to the right, holding back the left corner of the mouth. In this way a more direct view of the tonsils can be obtained than by looking straight into the mouth.

Observe in every case the condition of the little cleft in the antero-superior aspect of the tonsil, known as the supratonsillar cleft or fossa, the opening of which is often occluded by a small fold of mucous membrane. This fossa or cavity is often occupied by cheesy plugs, composed of epithelium, mucus, leucocytes, lime salts, and bacteria, which may form the starting point of inflammatory troubles.

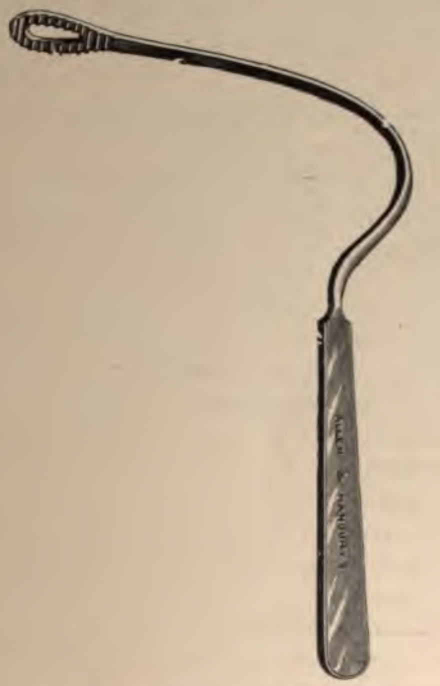
FIG. 3.—Fränkel's Tongue-Depressor.

Finally, examine carefully the posterior pharyngeal walls, observing the condition of the surface—whether dry and glazed, granular, or, it may be, ulcerated.

Muco-pus clinging to the posterior wall of the pharynx is indicative of nasal suppuration, and should be carefully noted.

When the pharynx of a patient lying on his back *in bed* is to be inspected the electric lamp is laid on the pillow beside the patient's left ear while the surgeon, wearing his forehead mirror, sits on the bed at the patient's right side and leans over him.

If the patient sits up in bed and turns his face towards the surgeon the rotation of the head renders the transverse process of the axis prominent in the side of the pharynx towards which the face is turned. Do not mistake the prominence of this region in that attitude for retropharyngeal abscess or an enlarged tonsil (Douglas Guthrie).

For examination of the naso-pharynx, see p. 220.

INSPECTION OF THE THROAT IN CHILDREN

This is always a difficult proceeding in a child with a tempestuous individuality, but by attention to the following instructions the performance will be robbed of its terrors alike for the mother and the doctor.

Seat the child in the mother's or the nurse's lap, not sideways, as is the natural mode, but with the back of the child against the chest of the nurse, so that it faces the doctor sitting in front. The nurse then separates her knees slightly, and as the child's legs slip in between them she clenches them together again. That prevents kicking, and preserves the integrity of the doctor's shins. Next, instruct the nurse to pass her right arm and hand —or, if the little patient is muscular and violent, both arms— round the child's chest outside his arms, gripping his hands while doing so. The patient is now held tight in a living vice, all save his head. To prevent it being shaken about, ask the nurse to put her spare hand, if she has one, round the forehead of the child, and to hold back the head firmly on her chest. If the nurse has no hand to spare, then the doctor can control the head with his left hand on the forehead, while he manipulates the teaspoon or other tongue-depressor with his right.

Driven thus to his last defences, the boy now clenches his teeth, breathes defiance through his nose, and awaits events. They need not detain him long. Pinch his nostrils together, so as to stop nasal breathing, and as he opens his mouth to gasp in some air, slip the tongue-depressor in over his tongue, and the game is up.

Some children can breathe through the mouth with the teeth clenched. In that case, pass your forefinger between the cheek and the teeth and back to the gums behind the last molar.

Insinuate the point of the finger between the jaws at this spot, and the mouth will open.

Once the mouth is open, it can be kept open indefinitely by pushing the lower lip over the lower incisors, so that if the child attempts to close his mouth, he will bite his own lip. This little manœuvre may, however, lead to a wound and some blood flowing, consequently it should not be adopted save as a last resource. In nearly all cases the mouth can be kept open by the depressor on the tongue, or, if necessary, by pushing back the depressor so as to tickle the fauces and induce gagging. This has the added advantage of bringing the tonsils fully into view.

The Base of the Tongue.—For a satisfactory examination of this region, which lies beyond the circumvallate papillæ and the foramen cæcum, the laryngeal mirror is required (see p. 80). The tongue here is attached laterally to the anterior pillars of the fauces and posteriorly to the epiglottis by the central glosso-epiglottic ligament—the frænum of the epiglottis —on either side of which are the valleculæ, or right and left glosso-epiglottic fossæ, which in turn are flanked by the right and left glosso-epiglottic ligaments. This area is supplied by branches of the right and left superior laryngeal nerves, the sensory nerves of the larynx, and it is so extremely sensitive that it has been named the "trigger-area" of the swallowing reflex, an appellation which bears its own significance. When the mouth is closed touching this area sets in operation the involuntary part of the mechanism of deglutition; when the mouth is wide open, the same stimulus induces the act of retching, that being the terminal act of vomiting, which is, of course, the reverse movement to deglutition. Consequently, in anæsthetizing the throat for examination or operation, the anæsthetic should be applied to this area first. Here the **Lingual Tonsil** is found. It appears normally as a number of gentle elevations on the surface of the tongue; but, like adenoid tissue elsewhere, it is subject to hypertrophy.

LOCAL ANÆSTHESIA

There is, perhaps, no other department of medical practice in which local anæsthesia is so frequently and widely employed as in oto-laryngology. And of all local anæsthetics none is so reliable and powerful as **Cocaine**, so that, in spite of its toxic properties, it still remains the local anæsthetic of everyday use.

The dose of cocaine hydrochloride by the mouth is officially set down to be gr. $\frac{1}{5}$ to gr. $\frac{1}{2}$, which is equivalent to from ♏ii to ♏v. of a 10 per cent. solution of the drug. But this quantity is habitually exceeded in making local applications to the mucous surfaces of the pharynx, larynx, and nose.

Moreover, the phenomena of *cocaine poisoning* may be excited by quite small quantities of the drug in susceptible subjects. Consequently, the laryngologist must always be on the alert for toxic symptoms so that he may intervene to check their full development as soon as they make their appearance.

The *symptoms* of acute cocaine poisoning are: a feeling of faintness with tingling and numbness of the extremities, associated with pallor of the face and lips; clammy perspiration; a rapid, compressible and sometimes fluttering pulse; sighing respiration; and dilated and fixed pupils. These asthenic phenomena may set in suddenly and progress rapidly to end in unconsciousness and sometimes even in death.

Prevention.—Difficulty of controlling the absorption of cocaine renders it unwise to use the drug hypodermically. When, therefore, anæsthesia by infiltration is called for, as for example in tracheotomy, one of its substitutes, such as eucaine or novocaine, should be employed.

When used as a local application for the induction of surface anæsthesia, cocaine should always be freshly combined with *adrenalin*, the ordinary strength for pharyngeal and laryngeal mucous surfaces being 10 per cent. sol. cocain. hydrochlor. with an equal quantity of sol. adrenalin (1–1000). This combination is both safer and more effective than cocaine alone; and that for two reasons. First of all, adrenalin by its vasoconstrictive action not only restricts the absorption of cocaine into the general circulation, but, by denuding the local nerve-endings of blood, freely exposes them to the action of the cocaine and thus intensifies its local effects. For this reason, less cocaine may be used. Secondly, adrenalin absorbed into the blood acts in the system as a physiological antidote to cocaine.

In making applications of the anæsthetic solution to the pharynx and larynx it is advisable to apply the mixture in a cotton-wool swab to the area to be anæsthetized very precisely, and the patient should be instructed not to swallow but to spit out any excess of liquid that may escape.

Emotional syncope, favouring and increasing, as it does, the constitutional effects of cocaine, should be countered by keeping the patient recumbent when that is possible, and this injunction applies with particular force to operations under cocaine anæsthesia.

By attention to these rules the risk of cocaine intoxication is reduced to a minimum, even when large amounts of the drug are used. (See p. 92.)

Treatment.—As soon as toxic phenomena appear the patient's head should be lowered; all constrictions about neck and waist should be loosened; and smelling salts or amyl nitrite given to inhale. Brünings extols an intravenous injection of 1 c. cm. (♏16 approx.) of sol. adrenalin (1–1000) in ten times its quantity of water.

For the ordinary general discomfort that so often follows minor operations and examinations under cocaine the best remedy is a cup of hot black coffee.

Substitutes for Cocaine.—As we have already remarked, cocaine should not be used for infiltration anæsthesia. For this purpose *eucaine* (4 per cent. solution in water) with a few drops of adrenalin solution (1–1000) is a highly satisfactory substitute.

As anæsthetics for mucous surfaces, neither *novocaine* nor eucaine is as effective as cocaine, but of the two perhaps novocaine is the more satisfactory. I have also found *stovaine* to be a very reliable local anæsthetic.

The Cocaine Habit.—The possibility of initiating the cocaine habit in our patients should not be forgotten. For this reason, it is better not to name the anæsthetic to the patient, and when patients come regularly for such minor manipulations as Eustachian catheterization the little operation should be performed without cocaine.

CHAPTER III

AFFECTIONS OF THE PHARYNX AND TONSILS

ANATOMICAL ABNORMALITIES

Perforations of the Anterior Pillars of the Fauces are not uncommon. They are often bilaterally symmetrical, and this feature, combined with an absence of scarring, has led to the belief that they must be of congenital origin. As against that view, there is the undoubted fact that Sir Robert Woods, of Dublin, and other reliable observers have seen them in process of formation in severe scarlatina. That this is probably the origin of all such perforations is supported by the circumstance that they have not, so far, been reported as occurring in young infants, and that the patients have or have had enlarged tonsils the swelling of which during the attack causes the perforation by pressure atrophy.

The condition gives rise to no symptom, and requires no treatment.

Bifid Uvula, which is a partial cleft palate, is also of no practical importance.

Perforation of the Palate, hard or soft, is syphilitic manifestation (see later, p. 614), and so also are the densely adherent and stellate scars occasionally seen in the throat.

Adhesion of the Soft Palate to the posterior pharyngeal wall, when associated with evidences of old ulceration and destruction of tissue, is usually syphilitic, but Vincent's Angina in its severer manifestations is said also to cause ulcerations as deep and as extensive as those of syphilis. It must be confessed, however, that the distinction between ulceration of the pharynx, due to syphilis, and that due to Vincent's Angina, is not always easy to make. (See p. 36.)

Adhesion of the Soft Palate to the posterior pharyngeal wall as a rare *sequela of scarlet fever,* is described, and the writer has reported one such case in which the adhesion was complete. In this variety, scarring is inconspicuous, or entirely absent, and the distortion of the pharynx characteristic of the syphilitic variety is absent.

The *symptoms* vary according as the adhesion is complete or partial.

If *complete*, the patient is unable to breathe through the nose, to smell, or to appreciate flavours, and the voice has the flat, toneless character of nasal obstruction.

If *partial*, the difficulty of nasal respiration is chiefly manifested in an inability to blow the nose freely, while mouth-breathing, especially at night, is habitual.

Diagnosis.—Inspection is sufficient to reveal the cause in the syphilitic adhesion, but in the scarlatinal type the obstruction is only to be discovered by naso-pharyngeal examination. The latter type must be differentiated from occlusion of the posterior choanæ. (See p. 235.)

Treatment.—The treatment of cicatricial occlusion of the naso-pharynx by surgical reconstitution of the cavity is extremely difficult. It is, no doubt, easy under an anæsthetic to re-form the cavity by a division of the tough adhesions which occupy it; but the difficulty lies in preventing their recurrence. For this a couple of sufficiently long drainage tubes may be passed in through the nose and out by the mouth, and retained in that position during the healing of the wound. Tying the loosened soft palate forward to the incisor teeth, and grafting are other devices which have been tried.

No operation should be performed at all in syphilitic cases unless the surgeon is sure that the original disease is entirely quiescent. Otherwise, he may light up new ulceration and leave the patient worse than before.

Large, **Pulsating Vessels** are occasionally visible on inspection on the posterior pharyngeal wall. They would probably deter one from operating, if seen, but adenoids have been removed without injuring such vessels.

Cases are known where a loop of the internal carotid has occupied the tonsillar region, and has been excised by the guillotine, with immediately fatal results.

An **elongated** and curved **styloid process** occasionally presents as a sharp bony process in the tonsillar region. It may cause pain, in which case it should be removed by cutting bone forceps after its exposure through an incision (Macleod Yearsley).

Congenital Shortness of the Hard Palate is a condition to which Brown Kelly in this country has drawn attention. It is rare, but not so rare as was at one time supposed. One can recognize it by the abnormally large gap which lies between the posterior surface of the soft palate, itself normal in size, and the posterior naso-pharyngeal wall. The subjects of this abnormality do not necessarily speak through the nose ; but they are liable to become deaf from post-nasal and Eustachian catarrh.

SORE THROAT (PHARYNGITIS AND TONSILLITIS)

General Consideration.—Acute pharyngitis may be the prodromal event of influenza, measles, scarlet fever, typhoid fever, or small-pox ; of the common catarrhal fever or " cold " ; of lacunar tonsillitis, or of peritonsillitis ; while it may be due to syphilis, or, it is said, to gout and rheumatism. It may also be the usher of the severer septic inflammations of the throat ; and, finally, drugs such as arsenic, iodide of potassium, and mercury may cause acute pharyngitis.

The pathological changes consist in hyperæmia and inflammation of the mucous lining of the pharynx, together with a varying amount of necrosis of the superficial epithelium, and altered mucous secretion.

The *symptoms* vary with the severity of the attack. In the milder cases a short initial stage of *malaise*, with some chilliness, seldom amounting to actual rigor, is followed by symptoms indicative of a certain amount of general infection—pyrexia, headache, muscular weakness, and loss of appetite. Locally, stiffness and soreness in the throat on swallowing are experienced, and signs of extension of the catarrhal process to the nasal passages or to the larynx are not uncommon. After a few days, the symptoms gradually subside, and the patient recovers.

In the severer cases the symptoms are more acute ; the throat is more painful, the fever higher, and the other constitutional phenomena more marked.

On examining the throat, the fauces, tonsils, and posterior pharyngeal wall will be seen to be uniformly and more or less deeply congested. The uvula is red and œdematous. If the tonsils seem to be the chief seat of the inflammation, we name the disease *tonsillitis* rather than pharyngitis.

Pharyngitis may be but one aspect of a general catarrhal infection, in which the nose, the larynx, the bronchi, and even the lungs participate.

Diagnosis.—Care should be exercised not to be satisfied with the diagnosis of simple pharyngitis until the course of the illness enables us to be quite sure that the "sore throat" is not due to measles, scarlet fever, diphtheria, or to one of the forms of the more serious septic pharyngitis.

Treatment can only be symptomatic until the cause has declared itself.

If there is much fever and malaise, the patient should go to bed, but the milder cases may remain ambulant.

Locally, the use of gargles or throat-paints relieves the sore rawness, especially if glycerine form the basis of the application. Steaming the throat (inhalation) with ♏v of carbolic acid in a pint of boiling water, also gives relief.

Externally, a wet compress of lint with a generous covering of gutta-percha tissue may be applied to the front of the throat, and held in position by a woollen bandage.

Internally, the patient's general discomfort may be mitigated by 10 grains of aspirin every four hours while we are awaiting the development of the symptoms.

Note.—As the bacteriological knowledge of the different clinical varieties of acute and subacute inflammations of the pharyngeal regions is still incomplete, it is impossible to make a strictly logical classification of these diseases.

The following is the arrangement we shall adopt:—

Acute Infective Pharyngitis ("Septic Sore Throat," "Septic Throat")

1. Acute Tonsillitis and Pharyngitis ("Hospital Sore Throat").
    - (a) Tonsillar Abscess;
    - (b) Peritonsillar Abscess;
    - (c) Adenoiditis;
    - (d) Lingual Tonsillitis.
2. Membranous Tonsillitis and Pharyngitis ("Membranous Sore Throat").
    - (a) Non-diphtheritic and "Septic," or Simple Membranous Tonsillo-pharyngitis;
    - (b) Vincent's Angina;
    - (c) Diphtheria.
3. Virulent "septic" (septicæmic) Pharyngo-tonsillitis (Gangrenous Sore Throat; or Acute Cellulitis; or Acute Oedema of the Pharynx, Larynx, etc.; or Ludwig's Angina)
4. The Pharyngitis of the Infectious Fevers.
5. Retro-pharyngeal Abscess.

ACUTE TONSILLITIS

is sometimes divided into the three varieties: (a) Catarrhal; (b) Lacunar or Follicular; (c). Parenchymatous; but these are in reality merely different stages in the same disease, and may be described together.

Etiology.—The tonsils may be looked upon as exposed lymphatic glands. Consequently, any septic invasion of the regions which they drain, the nasal cavities, the naso-pharynx, and the pharynx itself (exclusive of the tongue), may induce tonsillitis. Thus, acute tonsillitis is common after operations on the nose and in nasal accessory sinusitis. But there is no doubt, also, that the tonsils may become primarily infected, the causative organism entering, just as the tubercle bacillus does, by the pharyngeal surfaces and crypts of the organ. And this type of the disease is predisposed to by any cause lowering the general health, such as overwork, anxiety and exposure, as well as by local diseases, like pyorrhœa alveolaris and dental caries, which favour the presence of septic organisms in and about the throat, and by disease of the tonsils itself, which lowers the local resistance. Thus, recurrent tonsillitis is nearly always associated with more or less enlargement of the tonsils. But it should not be forgotten that the disease may attack small tonsils, and, indeed, that a septic infection of the tonsil region has been known to occur even after the tonsils has been enucleated.

The disease in epidemic form has been traced to infected milk, but sewer gas, and bad drains, at least in the patient's own residence, have probably no part in the causation of septic tonsillitis.

Acute rheumatism is an effect, not a cause of septic tonsillitis.

Bacteriology.—Acute tonsillitis is of microbic origin, and any of the pyogenic organisms may be grown from the surface exudate. The infection is usually mixed, strepto- and staphylococci being the commonest organisms found. But as all these may be recovered from the healthy mouth or throat, it must be admitted that the real bacteriological factor in the etiology of acute tonsillitis is still unknown.

Pathology.—The inflammation varies in incidence according as the disease affects chiefly the surface, or the crypts, or the substance of the gland. In any case, however, the crypts tend to become filled with inflammatory débris and exudate, and this material appearing at their orifices gives the characteristic "spotted" appearance to the inflamed gland. The body of the tonsil occasionally becomes the seat of abscess, by the coalescence of small pus foci, and in a proportion of cases the inflammation traverses the capsule and sets up a cellulitis in the pharyngeal connective tissue deep to the tonsil (peritonsillitis), which, leading to abscess formation, is the familiar peritonsillar abscess or quinsy.

Acute septic tonsillitis is accompanied or followed by acute rheumatism not infrequently; by acute endocarditis, chorea, and other evidences of the mitigated pyæmic affections. But genuine pyæmic abscesses do not form.

Symptoms.—The onset of the illness is apt to be sudden. After some shivering or a rigor, the temperature rises to from 100° to 103° F.; the pulse becomes rapid—100 to 120 per minute; there is severe muscular prostration; perspiration; anorexia; headache and constipation. Some nocturnal delirium may occur. The tongue is covered with a thick, yellowish fur, and is indented round the margins by the teeth. Pain and soreness in the throat, when complained of, lead to an examination, and the true cause of the attack is soon discovered. But it frequently happens that a day or two elapses before the patient feels any sore throat, and, indeed, the phenomena of rheumatic fever may actually develop without the patient making any complaint whatever about his throat, and the disease there is not discovered at all unless an examination is made.

This latency of throat trouble is specially common in children. Hence, the invariable clinical rule: **In all feverish attacks in children, examine the throat** as a matter of routine.

Latency also characterizes the tonsillitis following nasal operations.

In other cases, however, the pain and soreness in the throat are very distressing. Swallowing causes severe pain, and yet the patient is tormented by an uncontrollable inclination to swallow.

When the swelling of the tonsils is great, the respiration is impeded to the extent of compelling the patient to utilize his mouth in breathing, while the respiration is stertorous during sleep. As a matter of fact, however, the local pain and general misery combine to keep the patient awake, and the ability to get some sleep is often the first sign of approaching convalescence. The temperature runs a remittent course between 100° and 103° F., falling very gradually to the normal.

The voice is nasal. The head is stiffly held, and the patient may become a little deaf.

The most severe cases are those in which the interior of the tonsil becomes the seat of abscess, as in that case the local swelling and pain are greatest.

Examination shows an acute pharyngitis, in which the tonsils are specially involved, being swollen and projecting from behind the anterior pillar of the fauces. Their surface presents small, yellowish-white spots, which, in the severer cases, coalesce and give the appearance of a membrane. And, indeed. the exudate may at times be actually tough and membranous. As a rule, however, it is pultaceous and non-adherent. The lymphatic glands at the angle of the jaw are enlarged and tender.

Course.—The illness lasts from four or five days to a fortnight, excluding complications. The convalescence is tardy, and may be interrupted by relapses.

There is a surprising amount of muscular and constitutional weakness left, and several weeks must elapse before the last effects even of uncomplicated attacks disappear.

If the cause is allowed to remain, recurrent attacks are to be expected.

Diagnosis, as a rule, is easy. The attack is differentiated from diphtheria by the high temperature; by the absence of profound asthenia; and by the bacteriological examination. The last should never be omitted if membrane or suspicion of membrane be visible.

Tonsillitis, or what looks like simple lacunar tonsillitis, is frequent in scarlet fever. It may, indeed, be the only obvious sign of that disease. But scarlatinal tonsillitis is associated with a bright, vivid redness of the whole of the pharynx, and with a rapid pulse; while the rash, if present, is characteristic with its myriads of red points so close set as to seem like a diffused blush.

Prognosis is favourable so far as life is concerned, if the disease is one of simple septic tonsillitis.

Treatment.—The patient is kept in bed and is given whatever food he is able to swallow.

It is a usual rule to give internally salicylate of soda, in 10 grain doses every four hours, or acetyl-salicylic acid or aspirin (gr. x). But it is questionable whether these have anything other than a sudorific effect.

The tonsillitis following nasal operations may be aborted in twenty-four hours by a dose of antistreptococcus serum (25 ccs.).

Perhaps the most useful topical agent is sol. hydrogen peroxide (10 vols. per cent.), as a spray or gargle, or what is better, as a direct application to the interior of the crypts on a cotton-tipped probe. It cleanses the crypts, and, reducing the septic absorption, materially improves the constitutional symptoms.

In children, liq. ferr. perchlor. ♏vi to x with glycerin every four hours is a favourite remedy internally.

In the milder cases, treatment is often very successful. In the severer cases, on the other hand, the disease seems to pursue its own self-limiting course independent of our therapeutic efforts.

The subsequent care of the patient involves a watch being kept for acute rheumatism, endocarditis, albuminuria, and paralysis. The last appearing after a sore throat of any kind, means that the disease has been diphtheria.

If lacunar tonsillitis attacks a patient more than once, the tonsils should be enucleated.

Tonsillar Abscess.—Abscess in the body of the tonsil is an occasional result of septic tonsillitis. Its presence is denoted by swelling of the body of one tonsil persisting after the acute stage of the tonsillitis is over.

It may escape notice if it points and breaks on the posterior or inferior aspect of the gland. If discovered, the abscess should be opened by an incision.

PERITONSILLAR ABSCESS (QUINSY)

Etiology and Pathology.—Peritonsillar abscess develops in the cellular tissue above and in front of the tonsil, and is due to a septic cellulitis of this region. The infection is secondary to sepsis, which may be latent, of the tonsil itself, especially of the supra-tonsillar fossa. As the abscess develops in the palato-pharyngeal cellular tissue, it pushes the tonsil itself downwards and backwards, and so out of sight of the examining eye. Left to itself, the abscess sooner or later bursts, usually through the anterior pillar of the fauces.

Symptoms.—The onset is sudden, unless there has been a previous attack of tonsillitis.

Unilateral pain shooting up into the ear draws attention to the throat, and even in its earliest stages the pain is so characteristic that the patient, if he has ever had a quinsy before, at once realizes that he is in for another. The pain and infiltration render swallowing both painful and difficult. This is accompanied by the usual signs of a severe septic focal inflammation: malaise, asthenia, and pyrexia (about 102° F.), which, with the difficulty in swallowing, cause great misery. As the disease progresses and pus forms, the constitutional phenomena undergo some mitigation, and the pain becomes a little easier, but genuine relief is only obtained—though then with pleasing promptitude—when the pus is evacuated.

The aspect of the patient is characteristic. He sits up in bed, with his head inclined to the side of the disease, and usually supported by the corresponding hand. The face is pallid, the mouth is half open; the teeth and breath are foul; and his expression is that of patient misery. He holds the head and neck stiff, and, when he turns to look, does so from the shoulders.

Examination.—The mouth can only be partially opened, and examination is difficult, but it may be facilitated by getting the patient to grin, so as to admit more light into the mouth. There is great swelling of the tonsillar region, faucial pillar, and soft palate on the affected side. The tonsil is out of the line of view, and need not be looked for. Fluctuation cannot be determined by palpation. The inflammation affects the whole of the throat, and the uvula is swollen and semi-translucent from œdema. The tongue also is swollen, tooth-indented, and covered with thick fur.

The corresponding side of the neck is swollen and tender, and the upper carotid group of the lymphatics is enlarged.

Course.—After from four to ten days of pain, insomnia, dysphagia, and anorexia, the abscess bursts, often during sleep, with immediate relief to all the symptoms, and in a few days the patient resumes his normal life. But a second attack, on the other side, is by no means uncommon.

Treatment.—Attempts are still made to abort quinsy in its earliest hours by ♏i Tc. aconit. every hour.

General.—The patient may be kept in bed, but is often easier sitting up in a chair. His diet consists in whatever he can get down, such as raw eggs, milk, custards, and slops generally. Any treatment which necessitates swallowing, adds to the misery, and therefore all internal remedies by the mouth should be avoided. The same is true of local treatment by gargles, sprays, and douches, with the solitary exception of a warm solution of sod. bicarbon. which cleans the throat by " cutting " the mucus.

As early as the third day, if not before, an attempt should be made to open the abscess. The most prominent area of the swollen soft palate is the seat of election. The spot lies at the intersection of two lines, one tangential to the curve of the anterior faucial pillar, the other drawn through the base of the uvula (Fig. 4). After anæsthetizing the area with 10 per cent. solution of cocaine, the mucous membrane is incised with a long-handled, short-bladed knife. Then a closed pair of dressing-forceps is inserted into the incision, pushed forcibly backwards into the main mass of the swelling, opened, and withdrawn open. If the abscess cavity is penetrated, a gush of pus follows the withdrawal of the instrument. If pus is not encountered, a further search may be made for it with a grooved director.

The operation is painful, and the patient's head should be held firmly by a nurse. Failing skilled assistance, seat the patient with the back of his head to a wall so that he cannot jerk back out of reach when the forceps are being inserted. A general anæsthetic is dangerous, and should not be employed.

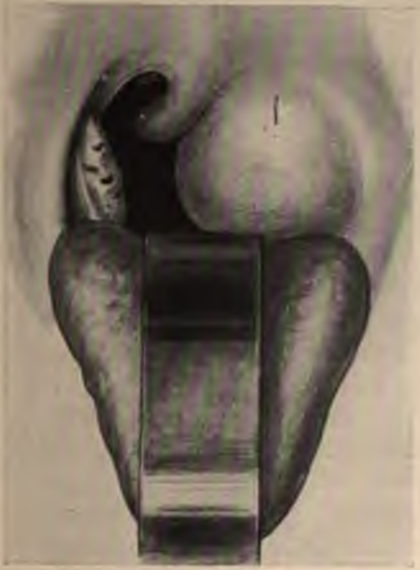

FIG. 4.—Site of the Incision for Peritonsillar Abscess.

Even when pus is not found, the patient experiences relief from the operation. But when it is found, the relief is instantaneous and complete. A simple antiseptic gargle suffices to complete the cure.

But a peritonsillar abscess is sooner or later followed by another on the same side unless the chronically diseased tonsils (which are the cause) are eradicated. Such tonsils may be quite small and inconspicuous on examination; but their size is no clue to their virulence.

Adenoiditis.—The frequently recurring "colds" to which children with adenoids are subject are undoubtedly due to attacks of septic inflammation affecting the adenoid masses. Abscess formation is rare.

The treatment consists in the removal of the adenoids. (See p. 377.)

Lingual Tonsillitis is a not uncommon accompaniment of septic lacunar tonsillitis The laryngeal mirror shows the red and inflamed masses studded with white spots. The treatment is the same as that for lacunar tonsillitis.

Abscess of the Lingual Tonsil occasionally follows septic inflammation of this area; or it may be due to puncture by a sharp foreign body such as a fishbone; dental abscess also may burrow in this region.

The *symptoms* are: severe pain with dysphagia, and sometimes a sensation of choking, or actual dyspnœa from obstruction by the inflamed mass. Constitutional signs of fever, malaise, and so on are also to be looked for. The mouth can be opened, but protrusion of the tongue is painful and impeded.

The diagnosis is made by the laryngeal mirror, which shows a bulky, rounded, red swelling at the base of the tongue, more prominent on one side than on the other. The epiglottis may only be seen with difficulty; it is frequently œdematous; and it is generally impossible to see into the larynx. The picture may be difficult to interpret unless one is careful to follow the contour of the swelling *forward* when it can be seen to merge into and become continuous with the dorsum of the tongue.

Diagnosis.—Abscess of the lingual tonsil is differentiated from simple hypertrophy, or from a lingual thyroid tumour by the pain, temperature, and signs of inflammation.

Treatment.—As soon as it is diagnosed, the abscess should be opened under cocaine anæsthesia. The tongue is held out by an assistant, and, guided by the laryngeal mirror, a knife with a long handle is carried to the back of the tongue, and plunged into the abscess. The little operation is not easy to perform, as the knife tends to glide off the slippery, resilient surface. A general anæsthetic should not be employed.

MEMBRANOUS TONSILLITIS AND PHARYNGITIS

The formation of a false membrane in the throat may be due to infection by (1) streptococcus; (2) more rarely staphylococcus; (3) the spirillum and fusiform bacillus of Vincent; (4) the Klebs-Loeffler bacillus (diphtheria).

In the septic (streptococcal and staphylococcal) varieties, the membrane forms on the tonsils first, and in the majority of cases it remains limited to them.

Symptoms.—There is considerable sore throat aggravated on swallowing, with high fever (102° to 104° F.), headache and prostration. The tongue is coated with a thick, white fur, and the glands at the angles of the jaw are enlarged and tender.

On inspecting the throat, the whole pharynx is seen to be generally congested; the tonsils are enlarged, inflamed, and invested, more or less, with yellowish, false membrane, which may also extend on to the faucial pillars and soft palate. The breath is fœtid.

The illness lasts for a week or ten days, running a course very similar to that of lacunar tonsillitis, and terminating in recovery almost invariably. Exceptionally, death may result in weakly children or adults.

The *diagnosis* raises the all-important question of diphtheria.

Clinically, the septic throat is more painful, the fever is higher and more prolonged, the pulse is faster, and, in short, all the reactionary phenomena are more lively, and the patient does not look so ill as in the graver disease.

Locally, the false membrane is as a rule less tough and less closely adherent than that of diphtheria, and the underlying surface, not being ulcerated, does not bleed when the membrane is artificially separated.

But these distinctions are merely suggestive. There is only one reliable method of differentiating between the septic sore throat and diphtheria, and that is by bacteriological examination. In no case should this be omitted or postponed.

Treatment.—Until the diagnosis is established, the case should be treated as one of diphtheria, the patient being isolated, and a full dose of anti-diphtheria serum given at once. Otherwise, the treatment of simple membranous pharyngitis is the same as that for acute septic tonsillitis and pharyngitis (q.v.).

VINCENT'S ANGINA

Etiology.—On the ulcers of this disease a fusiform bacillus, together with the spirochæta dentium, exists in great numbers. Hence it has been assumed that the ulceration is produced by the action of these organisms. This may be, and probably is, a correct assumption, but it has not yet been proved.

These organisms are normally found in the mouth, particularly around and in any septic focus, such as a carious tooth or pyorrhœa alveolaris. They have also been found in cervical abscess, in ear discharges, and even as far afield as in brain abscess (Wyatt Wingrave), and in fœtid bronchitis. In these conditions also the significance of the organism is not clear.

The *fusiform bacillus* of Vincent is a long organism with tapering ends; it is Gram negative, and can be cultivated in media to which blood serum has been added. The spirillum (*spirochæta dentium*) is long, thin and disposes itself in irregular spirals or lashes. Wingrave has suggested that these are two modifications of one and the same organism.

Vincent's Angina has recently been very widely disseminated among soldiers. It is feebly contagious.

Symptoms.—The disease runs a sub-acute or chronic course. The temperature may be normal throughout, and, in any case, rarely exceeds 101° F. The local symptoms are also mild; the soreness is slight, and the chief complaint is of the fœtor, and that is, indeed, objectionable.

On examination in the earliest stage of the disease, the tissue at the site of infection has a homogeneous grey look like india-rubber. Later, this breaks down into an ulcer with abrupt edges and red, irregular floor, devoid of membrane or secretion. The disease usually attacks one tonsil only to begin with. At a still later stage, the ulcer is covered with a foul dark-grey membrane, easily removed, and pultaceous in consistency. The underlying surface shows irregular granulations which bleed when touched. The ulceration, although usually unilateral and limited to the tonsil, occasionally goes on to affect other parts of the pharynx, and may even, it is said, lead to considerable destruction. Fatal cases are encountered, but recovery is the rule in from seven days to two months. The cervical glands are enlarged, and the neck itself is often puffy and swollen.

Diagnosis.—The disease is distinguished from diphtheria by bacteriological examination.

From tertiary syphilis the distinction is hard to make, inasmuch as its appearance closely resembles the tertiary ulceration; secondly, the Wassermann reaction is said to be frequently positive in Vincent's Angina; and finally, both diseases are amenable to the action of potassium iodide or to salvarsan internally. The diagnosis, therefore, will turn upon the presence or absence of other syphilitic phenomena.

We ought to add, however, that many reliable observers hold that the Wassermann reaction in cases of Vincent's Angina is never positive unless true syphilis is also present in the system (Taylor and McKinstry).

Treatment.—If possible the surgeon should himself cleanse the ulcer by picking off the membrane, and by spraying its surface with hydrogen peroxide (10 vols. per cent. solution). After mopping it dry, Tc. Iodi. should be accurately applied on a swab. Wingrave recommends trikresol (5 per cent. sol. in alcohol), or lysoform (25 per cent. in aq.). In the severer cases, salvarsan in solution may be applied locally, or it may be administered intravenously as in syphilis.

DIPHTHERIA

Diphtheria is due to the local growth in a virulent form of the Klebs-Loeffler bacillus. After inducing simple hyperæmia, the toxins of the bacillus cause necrosis of the epithelial layers of the mucosa, and thus produce the false membrane from which the disease gets its name.

The infection is conveyed by direct contact or indirectly through fomites. Cats and other domestic animals may also be sources of infection. Epidemics spring up from infected milk, and from " carriers," human or animal.

Incubation is short, from two to eight days.

Symptoms.—At first the fauces are simply reddened, and no hint of the true nature of the malady is afforded by the local appearances. But even at this stage there may be considerable general disturbance, shown in weakness, pyrexia, and increased pulse-rate; while in children, sickness and vomiting frequently occur.

In a day or two the throat gradually assumes the typical aspect of diphtheria, the membrane forming as a tough greyish-white or white pellicle on the tonsils or uvula, and gradually spreading to invest, according to the severity of the case, more or less of the mucous surfaces of the soft palate, pharynx, larynx, and nose.

For a day or two, there may be an apparent disproportion between the local signs and the constitutional disturbance. The temperature may be normal or subnormal, and the patient, for a time, appears to be easy and bright, although the membrane goes on extending, and the occurrence of secondary infection by septic organisms is manifested by the fœtid breath, the discharges from the mouth and nose, and the enlarged and tender lymph nodes behind the angles of the jaws. In these cases, death may suddenly occur from heart failure.

As a rule, however, general toxæmic symptoms are noticeable during the first day or so in the grey and earthy complexion, clammy skin, and compressible pulse, and the first sign of improvement, when it occurs, is remarked in these same general phenomena.

In cases which go on to a fatal issue by toxæmia, the prostration becomes more and more profound, the pulse feeble, irregular and flickering, and the end is usually rather sudden. In all the urine generally contains albumen.

Nasal Diphtheria.—Apart from the so-called chronic nasal diphtheria, diphtheria attacking the nose always leads to an increase in the general toxæmia. Its presence is indicated locally by an irritating discharge, which excoriates the alæ nasi and upper lip, and also, of course, by the presence of the membrane in the nose.

Chronic Nasal Diphtheria.—Membranous rhinitis, due to the Klebs-Loeffler bacillus, running a chronic, afebrile course, and without causing any general symptom whatever is an occasional experience. The symptoms consist in nasal obstruction, with irritating discharge, and a tendency to epistaxis. Examination will reveal the membrane, of a pale fawn colour, lying more upon the mucous surface than within it, thus contrasting with the membrane as it appears in the throat.

" *Carriers.*"—The diphtheria bacillus may persist for weeks or even months in the throat or nose of patients who have recovered from the attack. These people, as well as sufferers from chronic nasal diphtheria, are infectious to other individuals, and should be isolated. A " carrier " in a school may work considerable havoc until he is detected and removed.

Laryngeal Diphtheria.—Diphtheria of the larynx is one of the causes of " croup." When, as is usual, it comes on secondary to the pharyngeal attack, its occurrence is denoted by hoarseness, stridor, and the usual phenomena of laryngeal obstruction, proceeding, not infrequently, to serious interference with respiration, which, if unrelieved, may cause death from asphyxia. The tendency to obstruction is more marked in children than in adults; nevertheless, in adults, laryngeal diphtheria is a very fatal form of the disease (Rolleston).

Primary Laryngeal Diphtheria is denoted by hoarseness, cough, stridor, and by " croup " in children. Examination with the laryngoscope in children is difficult or impossible, and, in the absence of history of a foreign body, " croup " should therefore be treated as diphtheria until a swab is taken from the pharynx, and is reported to be free from diphtheria bacilli. The membrane may be coughed up.

Diphtheritic Paralysis may appear any time from the first to the sixth week, and lasts for weeks or months. The ocular muscles are those most frequently affected, and if it remain limited to them, the complication may escape notice, especially in children. After these, come the muscles of the soft palate. The paralysis varies in severity as in extent; when extensive, the muscles of the limbs and trunk being involved, death may result from paralysis of respiration, from heart failure, or from pneumonia.

Anæsthesia may also be produced.

The paralysis lasts for eight weeks or more. If the patient survives, the paralysis disappears entirely.

Diagnosis of Diphtheria.—In diphtheria the membrane is white or greyish, tough, and can be detached in flakes or pieces, laying bare an ulcerated, bleeding surface. In septic sore throat, the membrane is friable, and the underlying surface does not bleed when the membrane is removed. In diphtheria, the temperature tends to run a sub-febrile course; in septic sore throat it is high. In diphtheria, the general prostration is greater, and the signs of reaction, local and general, may be less marked than in the simple septic throat.

Septic sore throat is never followed by paralysis. In other words, if after a sore throat paralysis appears, then the infection must have been diphtheria. It occasionally happens that the sore-throat preceding the paralysis has been so mild that the patient has never been laid up at all.

In spite of those clinical differences, however, such appearances alone should never be altogether relied upon for a differential diagnosis, because diphtheritic tonsillitis or pharyngitis may be indistinguishable clinically from simple septic tonsillitis or pharyngitis. Thus it is a rule in all cases of sore throat to have recourse to bacteriological examination. Here we must utter a warning. If the bacteriological examination is negative for diphtheria, and yet the case looks like diphtheria, do not accept the bacteriological finding as conclusive. Have a second and even a third bacteriological examination made.

Further, all cases in which the clinical phenomena raise the question of diphtheria should be treated as diphtheria—isolated, and given diphtheria antitoxin, pending bacteriological examination.

In *taking a swab* of the throat for bacteriological examination, have the patient seated in the usual position for examination. Inspect the throat, and select in your mind the area best fitted for swabbing. For this, one makes for the place where the membrane seems to be most distinct and accessible. This is usually the tonsils.

Withdraw the swab from the sterile tube; moisten the wool in sterile water; pass it carefully into the throat, avoiding contact with teeth or tongue; apply it firmly to the selected area, and twist it round between finger and thumb. Then withdraw it as particularly as it was inserted, and replace it within the sterile tube. If a piece of membrane can be detached, and forwarded with the swab, so much the better.

Negative bacteriological reports in true diphtheria are usually due to an imperfect charging of the swab with the morbid material.

Swabbing children's throats is difficult. The child should be held as already described (p. 20), and the surgeon will find a Doyen's gag very useful.

Simple croup, in children, is clinically differentiated from diphtheritic croup by its sudden onset, either without any prodromal event, or in the course of an acute bronchial catarrh. If it does not disappear or materially improve in a few hours, treat the case as diphtheria.

Prognosis.—Diphtheria is more severe and fatal in children than in adults, and the more widespread the local lesion, the more serious are the constitutional phenomena. A soft, flickering pulse is of evil omen. The earlier the case is diagnosed and treated with antitoxin, the better is the outlook. After the fifth day, it is said, antitoxin is of comparatively little service.

Treatment.—Antitoxin should be given as soon as the diagnosis of diphtheria is made or feared. The rule is to start with 4,000 to 6,000 units, repeating the dose every twelve to twenty-four hours according to the symptoms, local and general. But in severe or delayed cases, this quantity may be greatly exceeded.

The injection is usually made in the flank. But in children, held on the nurse's or mother's knee, the back, between the scapulæ, is better. The area is painted with iodine-spirit solution, 5 per cent., and the injection made slowly into the subcutaneous cellular tissue. In the case of bulky injections, the needle may be left in the skin, and the syringe-barrel detached for re-filling. One should endeavour to distribute bulky injections over a wide area so as to minimize tension and pain.

[*Anaphylaxis* is the term used to describe the reactionary phenomena which occasionally follow the injection of foreign animal substances, such as horse-serum in the case of diphtheria antitoxin. These vary in severity from urticaria and asthma to sudden death. Typically they do not occur unless the patient has had a previous serum injection at least ten days before. But some subjects react dangerously to their first dose. (See later, Horse Asthma, p. 305.) The treatment of anaphylactic phenomena after their appearance is purely symptomatic.

Urticaria may be ignored. Collapse is countered by stimulation and position.]

Locally, the diphtheritic throat should be kept clean with hydrogen peroxide solution (5 or 10 vols. per cent.) in water, used as spray or gargle. And the ordinary diphtheritic throat requires no more than this.

Generally, dangerous cardiac symptoms are forestalled and met as they arise. If the pulse is feeble or irregular; if there is tendency to cold extremities or any other warning of collapse, the patient should be kept strictly flat, all sudden changes of position and all straining movements being forbidden.

Cardiac failure is treated by cardiac stimulants and by strychnine hypodermically.

Sir St. Clair Thomson recommends adrenalin (1-1,000) by mouth or hypodermically, ♏1 to 3, when paralysis is present, and has extended to pharynx or diaphragm, or when it threatens the heart.

Otherwise, paralysis is treated by rest in bed, by fresh air, and later by massage and electricity. It is also customary to employ strychnine in regular hypodermic doses.

(For a description of the affection of the throat in scarlet and other fevers, see p. 43.)

SEPTICAEMIC PHARYNGO-TONSILLITIS (ANGINA LUDOVICI; LUDWIG'S ANGINA; GANGRENOUS SORE THROAT; ACUTE CELLULITIS OR ACUTE ŒDEMA OF THE PHARYNX, LARYNX, &C.)

Under these various titles, of which "Ludwig's Angina" is the most popular, is described a rare and peculiarly virulent and fatal affection of the throat and cervical cellular tissue, characterized by intense and widespread local inflammation, and by profound and often rapidly fatal toxæmia.

Etiology.—Almost any of the familiar pyogenic organisms may be the cause, but the pneumococcus, and the streptococci are those most commonly found. The virulence of the disease may either be due to enhanced activity of the organism, or to a lowered tissue-resistance, or to both in combination.

Pathology.—The situation and extent of the throat and neck inflammation varies with each case; one showing more pharyngeal, another laryngeal, a third cervical inflammation. But in most, all three regions participate to a greater or less extent. In like manner the local lesion varies from an acute œdematous inflammation, with or without suppuration, to widespread gangrene. But severe as the intensity of the local lesion may be, it is the toxæmia which is most to be feared, as the fatal issue, when it occurs, is due to general septic intoxication.

It is generally considered that pathologically this disease is merely an intensified form of the simple septic throat. This may be true, but it is not the whole truth, since, to the clinician, the picture presented is profoundly different from that of simple septic throat.

Symptoms.—The symptoms are those of septic intoxication more or less severe, coupled with the local phenomena. The onset may be gradual, but it is often sudden, with a rigor. The height of the temperature varies, but it is always more or less raised.

Signs of grave blood-poisoning reveal themselves in a small, flickering pulse, cold extremities and general collapse, with tendency to death from heart failure.

Locally, the pharynx presents the appearance of severe pharyngitis with œdema of the uvula and soft palate. There is generally more or less laryngeal stridor which, on laryngoscopic examination, is seen to be due to œdema of the epiglottis, arytenoids, and parts about the entrance of the larynx. The inflammation generally spreads into the tissues of the neck, so that they become swollen and indurated, not pitting on pressure, and forming, in some, a collar-like swelling round the neck and in the submaxillary and sublingual regions.

There is remarkably little pain, but dysphagia is prominent from the muscular difficulty of swallowing, while severe and, indeed, dangerous dyspnœa from the laryngeal obstruction is a common symptom.

In the cases seen by the writer, the lymphatic glands have been swollen and tender.

The severity of the symptoms, general and local, serve to distinguish it from the commoner varieties of pharyngitis.

Prognosis.—The course of the disease is brief. Many cases swiftly recover, but others as swiftly become hopeless and die. The chief factor in forming the prognosis is the general condition. If the pulse keeps good, the mind cheerful, and the extremities warm, the chances are favourable. If, on the other hand, the pulse is rapid and small, the patient anxious and depressed, and the skin clammy and cold, the result is more likely to be bad.

One is apt in these cases to lay stress in one's mind upon the local difficulties of dyspnœa and dysphagia, and to think that when, for example, tracheotomy has been performed, the case will be out of danger. But that is a mistake. The chief peril lies in the absence of an effective constitutional reaction against the poison. The local difficulties can be overcome by art.

Treatment.—The profound toxæmia is met by antistreptococcus serum, 25 c.c., as soon as diagnosis is made, the dose being repeated every twenty-four hours if necessary, with general warmth and stimulants such as strychnine, ammonia, and brandy. Perchloride of iron is also useful.

Locally, warm fomentations of boric lint are applied to the swollen neck, and the patient steams the throat with boiling water, to which a few drops of carbolic acid may be added. In any case where the cervical swelling is a prominent feature in the case, free relief to the tension, or to the pus, if pus is present, should be given by incision through the skin, and the opening up of the deeper planes by blunt dissection wherever the induration seems greatest. The widespread œdema of these cases obliterates all landmarks, and necessitates careful dissection in the deeper cervical planes.

Tracheotomy should be performed at once when dyspnœa is severe. The trachea, in these cases, seems to lie very deep, but the dissection to reach it is easier than usual.

The operation, as well as the opening up of the deep cervical planes must be performed under local anæsthesia. The patients are too ill for a general anæsthetic.

EXANTHEMATOUS PHARYNGITIS

The Scarlet Fever Sore Throat.—In scarlet fever, the probable severity of the attack may be foretold in the early stages from the state of the throat.

Scarlatinal pharyngitis varies in degree from simple congestion to gangrenous inflammation. In the early days of the fever, the pharynx presents a characteristic bright red colour, uniformly diffused over the tonsils, fauces, palate, and posterior wall, but limited by the limits of the soft palate. In the severer cases, as time goes on, the congestion becomes more intense, there is some œdema of the uvula, and the pharynx is spotted with flakes of mucus. Later on, the posterior wall presents a glazed and polished appearance. Finally, these phenomena may become less marked, and the patient gets well, or they may be followed by the formation of sloughs of the pharynx or tonsils, and from the severe general disturbance characteristic of these cases, may terminate in death.

The tonsils in scarlet fever may be invested with a membrane, which may be diphtheritic, but is more frequently septic.

In scarlet fever, pressure atrophy of the anterior pillars of the fauces, which are stretched over the enlarged tonsils, may occur, leaving permanent fenestræ in the anterior pillars.

The slight angina which often attacks people exposed to scarlet fever infection, is, in all likelihood, scarlatinal in nature.

Oedema of the glottis, and even perichondritis of the cartilages of the larynx, may occur in scarlet fever, and the infection has a strong tendency to spread up the Eustachian tube into the middle ear, where it sets up a suppuration, which is frequently virulent. (See p. 469.)

There is also in scarlet fever a marked liability to the occurrence of acute septic infection of the lymph nodes in the neck which drain the pharyngeal and tonsillar areas. These glands undergo enlargement in all cases, and in some become the seat of acute abscess.

The local treatment is that of acute septic pharyngitis.

In **Measles** there is usually considerable pharyngitis, and the rash in the shape of red punctiform spots, sometimes with radiating streaks, appears on the soft and hard palate.

Koplik's spots have already been described. (See p. 15.)

RETROPHARYNGEAL ABSCESS

Retropharyngeal abscess may be acute or chronic. Both forms are commoner in children than in adults. Indeed, the acute type generally occurs in sucklings, and it is rare after the first year of life.

ACUTE RETROPHARYNGEAL ABSCESS

is due to septic infection of one of the chain of small post-pharyngeal lymph nodes which lie on either side of the middle line of the posterior wall of the pharynx. Any septic nasal disease in infancy may lead to its occurrence.

Symptoms.—The general symptoms are those caused by the formation of septic abscess. The signs which direct attention to the throat are: difficulty in swallowing, croupy and noisy breathing, and inability to breathe through the nose, a symptom which in very young infants always attracts the mother's attention. Fixation of the head from interference with the action of the anterior vertebral muscles, and perhaps from interference with the sterno-mastoid, is generally a marked feature.

On examination, a matter of difficulty in young infants with small mouths and the excessive mucous secretion common in this condition, bulging of the posterior pharyngeal wall will be seen. Palpation shows the bulging to be soft and perhaps fluctuant. As a rule, the swelling is lateral rather than central. There is concomitant general pharyngitis and œdema of the larynx may be taken for granted when croupy breathing is present.

If the abscess is not opened, it tends to burrow downwards into the mediastinum, with fatal results, or if it bursts spontaneously into the pharynx, it is likely to asphyxiate the infant.

Treatment.—The opening of these abscesses was at one time regarded as dangerous, but when properly performed, there is no risk.

An anæsthetic is not given. The child lies on its back on the nurse's knee as she sits opposite the surgeon.

The child's head is received by and grasped between the surgeon's knees. Under inspection by reflected light, for which in this inverted position the forehead mirror is turned round on the surgeon's forehead, the mouth is opened by passing the handle of a teaspoon, held by the surgeon in his left hand, along the child's tongue, where it acts as a tongue-depressor during the subsequent manipulations. The surgeon then takes in his right hand a straight, narrow-bladed bistoury, guarded to within half-an-inch of its point, and passes it into the child's mouth, with its cutting edge directed towards the palate. Having been guided safely past soft palate and uvula, it is plunged into the swelling, and the puncture thus made is converted into an incision by extension upwards; that is towards the palate.

All this is the work of a few seconds, but the important part of the operation is to follow. It consists in the nurse, when the surgeon gives the word, at once catching the child—it is usually a small infant—by the heels, and holding it upside-down until the abscess is emptied. The pus, unable to find its way into the larynx or trachea, flows freely from the mouth and nose. The nurse should be got to rehearse the manœuvre before the abscess is opened.

No after-treatment is necessary.

CHRONIC RETROPHARYNGEAL ABSCESS

is a cold abscess of tuberculous origin. There are two varieties, the distinction of which in diagnosis is of capital importance. These are, first, the glandular, and secondly, the spinal.

Chronic retropharyngeal abscess of *glandular* origin is also of two kinds. First, that due to tuberculosis of the retropharyngeal lymphatic glands proper; and secondly, that due to tuberculosis of the cervical lymphatic glands, usually of the deep carotid chain on one or other side.

Tuberculosis of the Retropharyngeal Lymphatic Glands, short of softening and formation of cold abscess, is by no means uncommon when cervical glands elsewhere in the neck are the seat of the same disease. And not infrequently in the retropharynx, as in the neck, they break down and form a cold abscess. Most cases are found in children, but the disease is not unknown in adults.

Symptoms.—It happens sometimes that no symptoms are complained of, and that this particular incidence of the disease is unsuspected until the throat is examined, as it ought to be, of course, in all cases of tuberculous cervical adenitis. (See p. 51.)

Attention is, however, generally drawn to the disease—in children, at all events—by symptoms resembling those caused by adenoids: impeded nasal respiration, with snoring at night.

On inspection, the posterior pharyngeal wall of one side is seen to be bulging forward. If the gland or glandular swelling has broken down, or is beginning to break down, more or less of the mass will feel soft to the touch. There is no pain and no rise of temperature. As a rule, tuberculosis of other glands co-exists.

Diagnosis.—The cold retropharyngeal abscess, from whatever lesion arising, is differentiated from acute abscess by the absence of fever and constitutional disturbance.

Tuberculous abscess of the retropharyngeal glands has to be distinguished from (a) tuberculous abscess of the carotid glands, extending inwards to the pharynx; and (b) from cold abscess due to caries of the upper cervical spine. The diagnosis is important from the point of view of treatment.

(a) When *tuberculous abscess of the carotid glands* is extensive, the collection may occupy a position extending from the outer planes of the neck into the retropharyngeal region. In such a case, in addition to fullness on one side of the posterior pharyngeal wall, there are signs of abscess on the same side of the neck, the collection coming to the surface under the skin usually posterior to the sterno-mastoid muscle.

Chronic Pharyngitis results from any long-continued irritation of the pharynx. Exposure to dusty atmospheres or irritating vapours ; excessive tobacco smoking or chewing ; dental caries and pyorrhœa ; alcoholism ; dyspepsia ; anæmia ; gout and rheumatism, are conditions which favour the production of the disorder, while occasionally it is set up by purulent nasal discharges, and not infrequently in adult life by the presence of septic tonsils.

Symptoms.—All forms of chronic pharyngitis are associated with discomfort in the throat—sometimes pain, more often soreness or rawness on swallowing, sometimes the feeling of a hair or a foreign body. An irritable hard cough often accompanies the paræsthesia, and the patient may complain of laryngeal disorders, such as hoarseness, quick tiring of the voice, etc.

On inspection, which is usually difficult by reason of the excessive sensitiveness of the throat, the whole of the pharynx—soft palate, uvula, tonsils, and posterior wall—is seen to be uniformly congested. The uvula is frequently elongated and œdematous. Enlarged mucous glands dot the palate and pharyngeal walls, and streaks of sticky mucus are to be seen flaked about the surface. At the same time, behind each posterior faucial pillar one can often see a thick, red hypertrophied band running up behind the soft palate, where, on endoscopy, it will often be seen to merge in a swollen posterior lip of the Eustachian orifice. This is sometimes called *pharyngitis hypertrophica lateralis.*

Granular Pharyngitis receives its name from the presence of small red granules of hypertrophied lymphoid clumps on the posterior pharyngeal wall. Granular pharyngitis is sometimes called " clergyman's sore throat," in which case the pharyngeal disorder is combined with laryngeal disability in the shape of vocal asthenia ; the need to " clear the voice " of frequently accumulating mucus ; and other irritating subjective phenomena, such as loud hawking and coughing in the morning.

Chronic pharyngitis in adult life is sometimes associated with the presence of septic tonsils, in which case the pharyngeal irritation may be secondary to tonsil infection.

Atrophic Pharyngitis or Pharyngitis Sicca is found in atrophic rhinitis and ozæna, and it is also induced when the inferior turbinals have been too much reduced in bulk by disease or operation.

The mucous membrane, especially of the posterior pharyngeal wall, is dry, glazed, and bright-red, bearing on its surface streaks of half-dried or wholly crusted mucus and muco-pus.

A variety of pharyngitis sicca occurs in diabetes, when it is associated with the dry, red, glazed tongue, characteristic of the later stages of that disease. A similar appearance is also presented in severe cases of scarlatinal pharyngitis towards the end of the first week.

Diagnosis.—The distinction between the different types of chronic pharyngitis, and the discovery of the cause in any particular case is not always easy.

Treatment.—Endeavours should be made to ascertain and treat the cause of the disease. Nasal disease, adenoids, septic tonsils and dental disease are among the commoner causes, and after them comes over-indulgence in tobacco, alcohol and rich food, all of which should be reduced.

Locally, while the cause is being treated, applications are useful by throat swabs of Liq. Perchlor. ʒi to ℥i of glycerine; Zinc Chloride grs., 20 to ℥i of water; or Argent. Nit., grs. 20 to ℥i of water.

Lozenges are favourite popular remedies for relieving the dryness and irritation of the throat.

Local hypertrophies and bands may be cauterized with the galvano-cautery.

ELONGATED UVULA

In chronic pharyngitis, from whatever cause arising, the uvula frequently undergoes lengthening, and may also become generally hypertrophied, broadened, and spade-like, in association with the inflammation and congestion of the soft palate.

The *symptoms* arising from the uvula are a sense of fullness and obstruction in the throat, and sometimes an incessant tickling cough, especially when the patient is lying down. The cough is obviously due to irritation of the glosso-epiglottic region, which is also known to proceed so far as to produce spasm of the glottis. In many cases the long uvula causes no symptoms whatever.

On examination, the uvula is seen to be resting on the tongue during quiet respiration, and on phonation, as it is tucked up by the azygos uvulæ muscle, the mucous membrane covering it is thrown into spiral folds.

Treatment, which is reserved for cases where it is really necessary, is by uvulotomy.

Under cocaine anæsthesia, the tip of the uvula is seized with a pair of uvula forceps and held, but not pulled forward, while with uvulotomy scissors, or with ordinary scissors curved on the

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flat, the tip and as much of the body as is redundant is cut off in an upward and backward direction, so as to leave the wound on the posterior surface. Hæmorrhage is slight. If it should continue, the raw surface may be touched with the galvano-cautery at a dull red heat.

A simple antiseptic gargle is all that is needed for after-treatment.

CHRONIC HYPERTROPHY OF THE TONSILS (ENLARGED TONSILS)

Etiology.—Enlarged tonsils are usually associated with adenoid vegetations in the naso-pharynx; indeed, in children it is rare to find enlarged tonsils without adenoids, while it is by no means unusual to find adenoids without enlarged tonsils. Moreover, adenoids of sufficient bulk to cause symptoms are found at an earlier age than enlarged tonsils. That being so, it is reasonable to assume that in addition to nasal infection, naso-pharyngeal infection also tends to cause enlargement of the tonsil. (See also p. 378.)

The tonsil is an exposed lymphatic gland, and its enlargement is almost certainly due to chronic infection. Further, the enlargement is not limited to the tonsil; it affects also the adjoining lymphatic glands. Of 170 cases of hypertrophied tonsils examined by the writer, in only three cases could no enlarged glands be felt in the neck; in twenty-two, the glands were noted as being "large," and in twenty-two, enlarged nodes were also felt in the posterior triangle of the neck. Those most frequently enlarged belong to the superior group of lymphatic glands of the deep cervical chain, and can be felt just behind the angle of the jaw. Thus the enlarged tonsil is merely one of a chain of hypertrophied masses of lymphoid tissue, a chain which, in childhood at least, usually includes the adenoid vegetations of the naso-pharynx.

If enlarged tonsils are not removed by operation, they in the course of time gradually become smaller, until by the time adult age is reached they have lost much of their bulk, although they often retain much of their power for evil. But this reduction in bulk is very irregular. In many cases they seem to get larger with increasing years, while in others they remain unaltered in size. In adult life the virulence of a tonsil is estimated, not by its bulk, but by its effects. In childhood, one judges more by the size, perhaps, when operation is being discussed, save, of course, in the case of tuberculosis (q.v.).

Symptoms.—In children, hypertrophied tonsils by their bulk and prominence add to the burden of adenoids by impeding buccal respiration just as adenoids impede nasal respiration. That this is the case was shown by the frequent disappearance of the symptom of snoring, which, in older times, before adenoids were discovered, followed when the tonsils alone were reduced.

Further, enlarged tonsils are septic tonsils, and so these patients, both children and adults, are subject to recurrent tonsillitis and pharyngitis; to acute rheumatism with its sequelæ, endocarditis and chorea; more rarely, to acute nephritis; and even, it is said, to appendicitis and other forms of gastro-intestinal sepsis. Lastly, enlarged or septic tonsils share, with carious teeth, the reputation of presenting an ever-open door for the admission of tubercle bacilli, which, passing to the neck, lead to glandular tuberculosis. (A. P. Mitchell.)

Consequently, patients with those diseases, as well as patients who are subject to recurrent attacks of "sore throat," "tonsillitis" and "quinsy," should be examined with the view of ascertaining whether or not the tonsils may be the cause of their ill-health.

On *inspection* hypertrophied tonsils are usually quite evident. If a tonsil projects from behind the anterior pillar of the fauces, its size should be regarded as abnormal. But it is frequent to find a large tonsil so hidden by a broadened or distended faucial pillar that it is entirely invisible. This is known as a "buried tonsil." But although the tonsil is thus hidden, its bulk so narrows the isthmus of the fauces that there need be no difficulty in recognizing its presence. Occasionally, however, the part of the tonsillar body most hypertrophied is the lower segment, and, as this lies below the level of the dorsum of the tongue, it may escape observation, not only in the ordinary routine examination of the throat, but what is more important, it is liable also to escape notice at the time of operation. Hence, the tongue should always be well depressed, and it is also a good plan to induce gagging or retching at some part of the examination, as this act turns the inner aspect of the gland obliquely towards the mouth, so that we get a complete view of the whole tonsil.

Diagnosis.—In adults the progressive enlargement of one tonsil raises the question of sarcoma (q.v.). And in lymphadenoma (Hodgkin's Disease) that disease frequently commences with an enlargement of the tonsils, bilateral in this case. But both in sarcoma and in lymphadenoma the surface of the gland is smooth, while the colour is paler than in simple hypertrophy. In any case of doubt, the microscope must be resorted to for diagnosis.

Treatment.—(1) When the tonsils are enlarged in a case of adenoids; (2) when they obstruct respiration, interfere with speech, or cause persistent cough; (3) when there is recurrent tonsillitis or quinsy; (4) when the tonsils are suspected of causing rheumatism, chronic pharyngitis or other disease, then they require treatment. The only treatment worthy of the name is operation, and in modern times the operation practised is total and entire removal of the gland—what is variously termed *enucleation* or *tonsillectomy.*

It should be noted also that when there is tuberculosis of the cervical glands, the tonsils must be entirely removed, and in this disease they should be removed whether they are enlarged or not.

THE REMOVAL OF TONSILS

may be performed by one of two methods, namely: first, *enucleation by the guillotine,* which is the operation of choice in children; and secondly, *tonsillectomy by dissection,* which is the operation of choice in adults.

Tuberculosis again furnishes an exception to this rule. In tuberculosis the tonsils should invariably be dissected out, whether the patient is young or old.

The *preparation* is the same for both operations, and indeed for all operations about the throat, naso-pharynx, nose, and larynx. Consequently, they will now be detailed once and for all.

Cleanse the mouth.—If there are any carious teeth; alveolar abscesses; or pyorrhœa alveolaris, the dentist must be employed to remove or minimize these complications. If this is not done, the operation wound in the throat is liable to become septic. After extraction of the teeth, the throat operation should not be performed until the dental wounds are healed.

After each meal for two or three days preceding operation, the patient is made to rinse the mouth, brush the teeth, and gargle the throat with a mild antiseptic mouth-wash, such as lysoform in Aq. 1 per cent.

The other preparatory measures are the same as those for any other surgical operation.

The Guillotine Operation as practised in children under puberty, will first be described.

Anæsthetic.—Open ether, chloroform or ethyl chloride. Whichever is employed, the patient is first of all anæsthetized, and then the operation is performed with the anæsthetic mask off. A second administration is seldom necessary, as the anæsthesia is sufficiently prolonged for both the tonsil and the adenoid operation.

Position.—In hospital practice with a large clientèle the sitting position enables the nursing staff to handle the cases quicker. A very stout chair is necessary, into which the patient is secured by a strap across the upper thighs. The surgeon, wearing his mirror, sits in front of the patient, as in the ordinary examination. The anæsthetist, standing behind the chair, leans over the patient's head, and administers the anæsthetic from that position. The tray of sterilized instruments lies on a table at one or other side of the surgeon. Kneeling on the floor by the patient on the side opposite to the instrument table is an assistant nurse, while the chief nurse looks after instruments and surgeon's requirements.

Performed in this manner, the operation can be accomplished with perfect efficiency, but " team-work " is called for, and unless all the co-workers are trained, it is much more satisfactory to operate on the patient lying flat on an operating or other suitable table.

In the same way, unless the anæsthetist is an expert, open ether or chloroform is preferable to ethyl chloride.

With the patient in the horizontal position many operators advise that the head should be lowered during operation so as to hinder the ingress of blood into the larynx and air-passages. The writer, on the other hand, always operates with the patient on an inclined plane, from head to feet, the head and shoulders being raised. In this position the hæmorrhage is less free.

With the patient lying on the table, the surgeon stands on the right ; the anæsthetist at the head ; opposite the surgeon is his assistant, surgeon or nurse ; while the lamp is placed at the corner of the table between anæsthetist and assistant. A fourth pair of hands takes charge of sponges and sponge-holders.

It is assumed that the strict ritual of aseptic surgery is followed. Operator and nurses wear sterilized gowns, gloves and masks.

Instruments.—Doyen's mouth-gag (Fig. 5) ; Fränkel's tongue-depressor (Fig. 3) ; half a dozen George's sponge-holders with marine sponges if procurable, otherwise gauze swabs are used; Heath's reinforced tonsil guillotine (Fig. 6); four pairs of artery-forceps, of large size. (For adenoid operations see p. 383.)

Manipulation.—The mouth is propped open with Doyen's gag. It is better to remove the *right* tonsil first. There are two methods of using the guillotine.

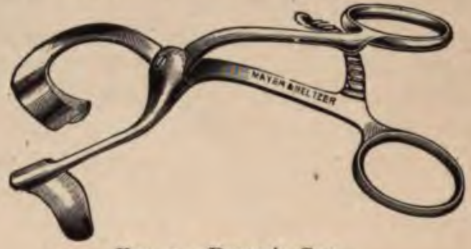


FIG. 5.—Doyen's Gag.

Method No. 1 (Fig. 8)—Holding the guillotine in the left hand, and with the handle turned to the patient's right, pass the eye of the instrument over the right tonsil. Carry the shaft of the instrument to the opposite angle of the patient's mouth, and press the tonsillar end of the instrument very firmly outwards. At the same time, pass the forefinger of the *right* hand into the patient's mouth, and apply it to the lower rim of the eye of the guillotine as it lies embracing the tonsil, and with this finger aid the forcible pressing of the guillotine around the tonsil. Then with the thumb of one or other hand, push home the guillotine blade. Turn the guillotine flat (i.e., with the plane of its broad surface parallel with the plane of the dorsum of the tongue), its handle being brought round so as to lie pointing downwards (i.e., towards

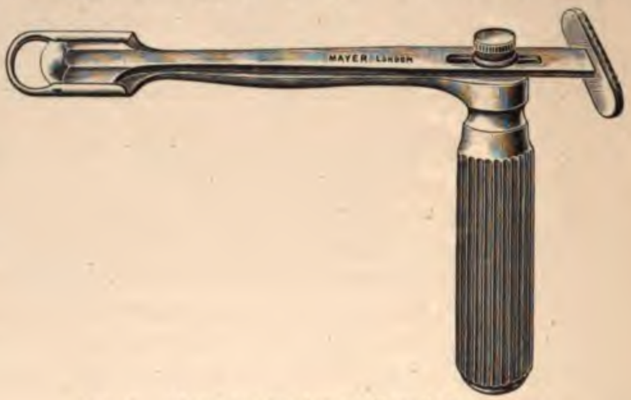


FIG. 6.—Heath's Reinforced Tonsil Guillotine.

the patient's chest), and withdraw from the mouth. The tonsil comes out fixed in the guillotine. Re-open the guillotine, and deal with the left tonsil in the same manner, substituting left for right in the manipulation.

Method No. 2.—To remove the *right* tonsil, grasp the guillotine in the right hand, with the handle pointing towards the patient's left. Pass the guillotine into the right tonsillar region from the left angle of the mouth. Pass the eye of the guillotine over the right tonsil. (*Note.*—The guillotine now occupies a position relative to the tonsil, the reverse of that described in No. 1.) Press the eye forcibly outwards, and while doing so apply the forefinger of the left hand against the anterior pillar of the fauces so as to push the buried part of the body of the tonsil into and through the eye of the guillotine. While this manœuvre is being accomplished, slowly push home, with a disengaged thumb, the blade of the guillotine. Finally, turn the guillotine flat as in method No. 1, but with the handle pointing upwards to the patient's forehead, and withdraw. Re-open the guillotine, and deal with the left tonsil in the same way, with left for right.

(1) (2)

FIG. 7.—Tonsil of Child Enucleated by Guillotine.
1. In the natural state, the tonsil capsule enclosing the gland like a hood.
2. In the extroverted state, with the gland substance extruded and the capsule reflected.
Compare with Fig. 8.

In *both methods* the object of the manipulation of the guillotine is to extrovert the mass of the tonsil body through the ring of the guillotine (see Fig. 8), and when this is successfully accomplished, the tonsil is removed in the extroverted state, entire, and with its capsule intact. (See Fig. 7.)

In *both methods* the object of the manipulation is facilitated if, after the guillotine is accurately and firmly applied, the instrument and its contained tonsil are pulled a little forward so as to cause the tonsillar region to ride, as it were, on the adjacent bone of the inferior maxilla.

Whichever method be adopted, the tonsil should immediately after removal be scrutinized to see that its capsular surface is smooth and intact. (See Fig. 7.) If it is not, the guillotine is re-introduced as before and the manipulations are repeated, and so on until the whole tonsil is removed.

Before proceeding to remove the second tonsil, sponge and inspect the wound made in removing the first. If the hæmorrhage is general and oozing, it may be ignored, and in children the same is true of a small spouting vessel.

Repeat the inspection after removing the second tonsil. (For the treatment of bleeding tonsil vessels, see p. 60.)

Adenoids, if present, are now removed. (See p. 383.)

Before returning the patient to bed make sure that the bleeding is stopped. If it is still continuing, the anæsthesia must be prolonged, and the bleeding points secured.

Difficulties.—The knack of guillotine enucleation can only be acquired by practice. There is a risk of button-holing, or of removing the anterior faucial pillar in pushing home the blade of the guillotine, but with complete extroversion this can be avoided.

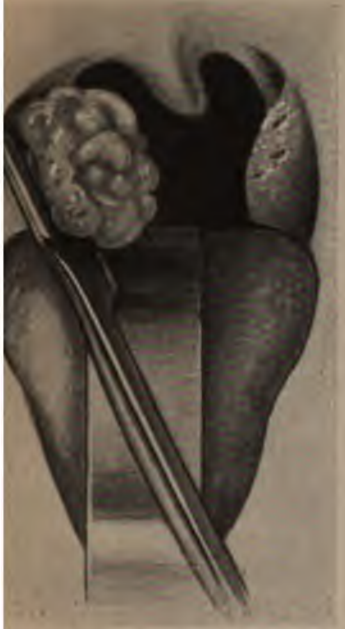
Extroversion is most difficult in old fibrous tonsils, and these, though commonest in adults, are sometimes also found in older children. The fibrous tonsil, if guillotined, is very liable to bleed.

After-treatment.—The patient is laid in bed, and kept during the first 24 hours on one side, with the face turned over and downwards (William Hill), pillows being adjusted to maintain the position. In this attitude blood from the throat tends to come into the mouth or nose, and to be ejected. If, on the other hand, the patient is left lying on the back he may bleed to death into his stomach.

Give instructions to the nurse to keep all vomited material during the first 24 hours, and to report if the patient is vomiting blood at frequent intervals. The surgeon should himself visit the patient not later than two hours after the operation, in order to intervene if hæmorrhage is proceeding.

During the first twenty-four hours the patient suffers considerable pain in the throat, especially on swallowing. But otherwise, the condition is not uncomfortable, and shock after operation is rare.

The antiseptic gargle should be continued, and the food restricted to slops until the patient can chew and swallow easily. He should be kept in bed for three days, and indoors for a week.



8.—Guillotine Enucleation of the Tonsil.
oversion through the eye of the otine is completed and the blade is closing.
Compare with Fig. 7.

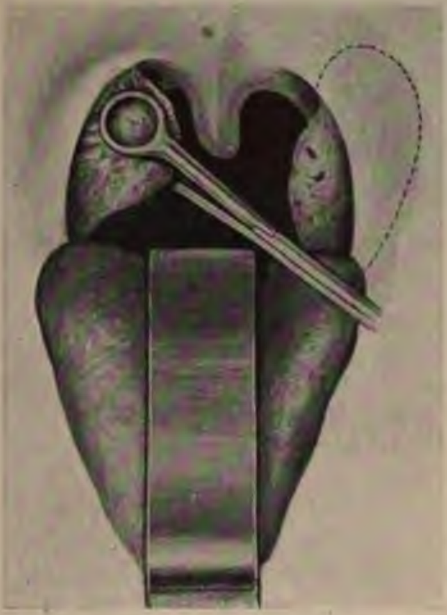
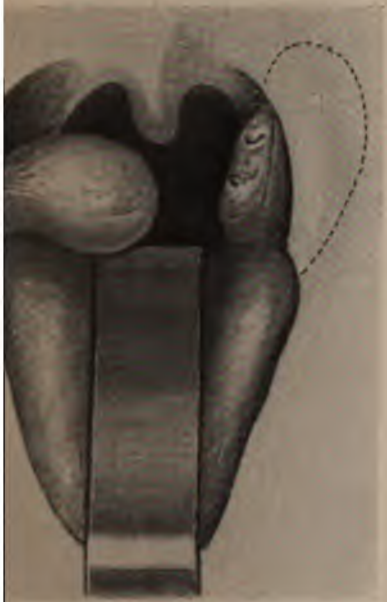


FIG. 9.—Tonsillectomy by Dissection. The incision is made parallel to the edge of the anterior pillar of the fauces through the pale line produced by the pressure of the buried part of the tonsil when pulled out with the forceps.



10.—Tonsillectomy by Dissection.
tonsil has been dissected out of its and is lying on the tongue, still attached by its lower pole.

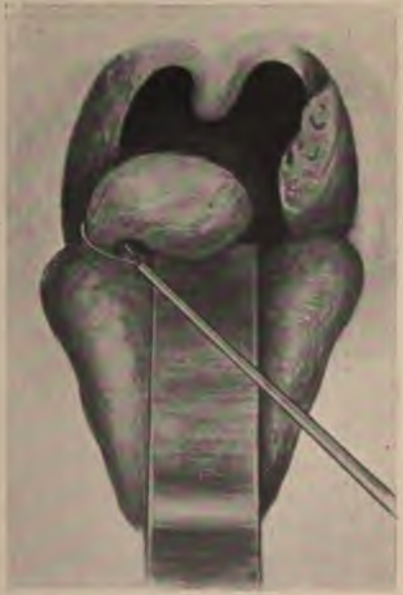


FIG. 11.—Tonsillectomy by Dissection. The snare passed to sever the last attachment.

Tonsillectomy by Dissection is always employed in adults. It is also necessary in children with cervical gland tuberculosis. It is a more severe operation than enucleation with the guillotine; the risks of sepsis are greater; and the subsequent scarring is more deforming to the fauces and palate. But it is preferred for adults because it is difficult to enucleate old tonsils with the guillotine, and because after puberty the guillotine operation is more often followed by severe hæmorrhage.

The preparation of the patient and the position at operation are the same as at the former operation.

Anæsthesia.—Prolonged anæsthesia, in the surgical stage, is necessary, and for this reason ether with chloroform is usually employed. The administration should be preceded in adults by gr. $\frac{1}{6}$ morph. hydrochlor. with gr. $\frac{1}{100}$ atropin hypodermically, in order to calm the patient and to reduce the secretion of saliva and mucus.

In adults, the tonsils may be removed under *local anæsthesia*. After the pharynx has been anæsthetized (pp. 22 and 92), the faucial pillars and the body of the tonsil are infiltrated with 5 per cent. sol. novocaine.

Manipulation.—The mouth is widely opened with a Doyen's gag. A stitch of silk is passed through the tongue, and left in, the ends of the thread being grasped by a pair of pressure-forceps. These are given to an assistant to hold, and he, by their means, keeps the tongue out of the mouth during the course of the operation.

The face is turned in such a way that the head lies more or less on its right side while the right tonsil is being operated on, and towards the left when the left tonsil is being operated on.

The tonsil is seized by Seccombe Hett's forceps, and pulled towards the opposite angle of the mouth. This makes the buried body of the tonsil bulge out the anterior faucial pillar (see Fig. 9). With a sharp scalpel an incision is made through this pillar on to the bulge as near to the edge of the pillar as possible; in length extending from the apex of the buried tonsil almost to the level of the tongue; in depth, cutting through the mucous membrane and submucous tissue, and *no deeper*. This reveals the capsule of the tonsil at its buried upper and anterior part. With a blunt tonsil dissector, or with a pair of curved scissors, closed, the capsule bearing its tonsil is rapidly freed from its loose attachments until the gland can be dislocated out from its bed on to the tongue (see Fig. 10). In this act it may be necessary to snip with the scissors the connection of the gland with the upper part of the posterior pillar of the fauces. The tonsil is now attached by its lower pole only, but as this attachment is firm, and, indeed, partly composed of muscle fibres from the superior constrictor of the pharynx, it cannot be got through by blunt dissection.

At this stage the guillotine may be threaded over the partially detached tonsil, and this last band of union cut with the blade of the guillotine. But the author advises Lermoyez's wire snare (Fig. 13) for the final act of severance, as it is less liable to be followed by bleeding (Fig. 11). The tonsil forceps are first of all passed through the loop of the snare, and both instruments are conveyed to the pharynx. The forceps grasp the semi-detached tonsil, and pull it through the loop of snare-wire, which is thereupon tightened. See that the uvula is not included in the wire-loop.

Take care before giving the final turn to the snare that the strangled tonsil is caught by a pair of forceps—Tilley's forceps are useful at this stage—to prevent the severed gland being lost in the pharynx. After the tonsil is thus removed, mop the raw surface left with a sponge, and look for bleeding points. (For bleeding, see later.)

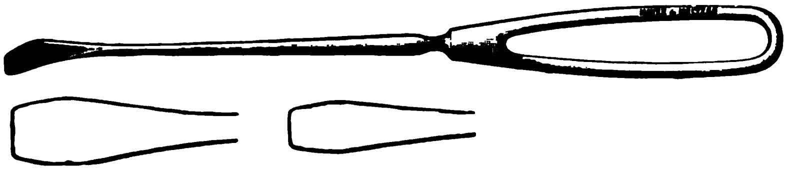


Fig. 12.—The Author's Tonsil Knife.

The bleeding stopped or negligible, turn to the other tonsil, and deal with it likewise.

Difficulties.—This is not an easy operation, and should not be undertaken lightly. The inexpert are prone either to remove too little or too much, and the risks from hæmorrhage are considerable.

It is a curious fact that pedunculated movable tonsils are more difficult to dissect out than buried tonsils are. In the case of the former the capsule of the upper part of the tonsil may be most easily reached and exposed by first of all inserting the author's tonsil-separator (Fig. 12) between the anterior pillar of the fauces and the body of the gland, sweeping it up and down so as to sever the attachment of the tonsil to the pillar. This manipulation is most easily carried out if the tonsil is not grasped and drawn out of its bed by forceps, at all events until the separation has been effected.

In this modification of the operation the separation of tonsil from pillar by means of the separator does away with the necessity of making the incision described above through the anterior pillar of the fauces.

Hæmorrhage.—The ordinary capillary and venous bleeding from the operation-wound can usually be stopped by sponge-pressure continued for several minutes. Some operators apply chemical styptics such as zinc chloride (grs. 30 to ℥i of water); tannic acid; liquor ferri perchlor. and so on. (Adrenalin should not be employed for this purpose as reactionary hæmorrhage may set in after its use, and may be very difficult of control.)

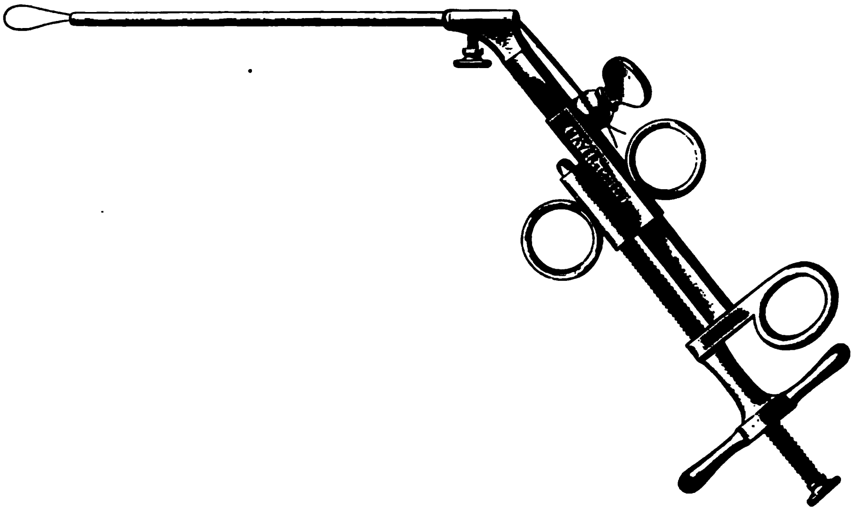


Fig. 13.—Lermoyez Tonsil Snare. (Wire with No. 9 or 10; English gauge.)

If, after the pressure has been continued for a few minutes, inspection shows that the hæmorrhage has not come to a standstill, search should at once be made for the bleeding vessel or vessels. Bleeding from such vessels, whether they be arteries or veins, is apt to be mistaken for general capillary oozing.

Generally, the bleeding point is located on the posterior aspect of the anterior faucial pillar. Take a pair of forceps, therefore, catch the anterior pillar, turn its raw posterior surface round, and you will see the bleeding point. It may be caught now by pressure forceps. If successful, the result is at once manifest, for the tonsil fossa becomes dry and remains dry. The vessel so caught can be ligatured; but if this is difficult, the forceps may be left on, until the second tonsil is removed. Then, when the bleeding from the second operation wound has stopped, return to the first, and remove the forceps. Often the hæmorrhage will be found to have ceased. If not, re-apply the forceps and either ligature the vessel or leave the forceps *in situ* for several hours.

The forceps recommended are a large pair of *straight* pressure forceps, or tongue or sponge forceps (George's pattern) used as pressure forceps; and stout catgut fresh from its spirit bath is more easy to manipulate than silk as a ligature.

Make sure that all tonsil bleeding is stopped before the patient leaves the operating table.

Sutures.—The upper area of the wound after dissection reaches high into the soft palate, and can be closed by catgut sutures passed by means of the author's needle. By doing so subsequent cicatricial deformity is reduced.

Reactionary Hæmorrhage.—As a rule what is called reactionary hæmorrhage is merely hæmorrhage prolonged after the operation ; that is to say, hæmorrhage which the surgeon has failed to check at the operation. It may be, perhaps, sometimes difficult to make sure at the operation that hæmorrhage has actually ceased.

At all events, if not discovered until after the patient has come round from the anæsthetic, ice should be given to suck, and the patient's head and shoulders raised. If this fails, we have to decide, when both tonsils and adenoids have been removed, which area it is that is bleeding. Hold the patient's head forward—adenoid bleeding comes from the nose, tonsil bleeding from the mouth.

If there is tonsillar hæmorrhage, assume the forehead mirror, fix up the light, insert a Doyen's gag into the mouth, and inspect the throat. If one tonsillar fossa contains a clot, that is the culprit. Otherwise the bleeding will be visible.

At this stage, the hæmorrhage is most readily checked by Watson Williams' clamp firmly and accurately applied, and left on for three or four hours, the discomfort of its presence being lessened by gr. $\frac{1}{4}$ to $\frac{1}{2}$ Morph. Hydrochlor. hypodermically.

True secondary hæmorrhage may appear in septic wounds a week or ten days after operation. If severe, it may be controlled by the clamps.

For uncontrollable hæmorrhage from the tonsil wound area the faucial pillars may be firmly sutured over a packing of gauze laid in the bed of the tonsil. Another plan is to cauterize the wound with the diathermy terminal (see p. 65) or with the galvano-cautery. Failing such local measures, the external carotid artery should be ligatured.

After-treatment is the same as that for removal of the tonsils by the guillotine.

Results are, as a rule, excellent. Enucleated tonsils do not grow again. But it is impossible to eradicate lymphatic gland tissue, and later on small lymphoid groups may make their appearance in the faucial pillars, and may even show the phenomena of tonsillitis! But as a rule there is no further trouble.

Not infrequently, considerable cicatricial deformity follows the healing of the wound. It can be minimized if, at the operation, the tonsil is thoroughly separated well down to its lower pole before applying the snare, so that the pillars of the fauces are not included in the wire-loop (E. D. D. Davis).

The removal of tonsils is sometimes followed by weakening of the voice for a few weeks or months after operation. This may be a disconcerting circumstance if the patient is a professional voice-user, but it is not permanent. The same remark applies to "speaking through the nose," a symptom by no means infrequent after tonsillectomy both in children and in adults. It disappears in about three months.

Tonsillolith.—Calcareous concretions are occasionally found in the tonsils, either as small discrete bodies embedded in the crypts or as large single calculi, which may reach a great size (C. H. Hayton). They are seldom discovered until after removal of the tonsil, but sometimes they give rise to pain and abscess. Cartilaginous and bony nodules are occasionally found in the tonsil.

Fish-bones or other sharp **foreign bodies** occasionally become fixed in the faucial tonsil. But they are more frequently found in the lingual tonsil.

If a foreign body in the throat is suspected by the patient, examine the throat carefully, and, if it is discovered, remove it with suitable forceps. But it will often happen that no foreign body will be found, the sensation being due to the slight traumatism produced by the foreign body while being swallowed. (See also pp. 164 and 209.)

NEOPLASMS OF THE PHARYNX

Papilloma is not infrequent on the edges or surface of the faucial pillars, soft palate and uvula. The growth is easily recognized by its rough, warty surface, and may be destroyed by an application of lunar caustic or by removal with scissors.

Angiomata are met with involving the soft palate and uvula. They may be treated by means of electrolysis or by diathermy puncture.

CANCER OF THE PHARYNX

Epithelioma.—*Etiology.*—Epithelioma of the pharynx is favoured by old syphilitic scars; leukoplakia of the palate; the habitual ingestion of very hot liquids; the presence of dental caries and pyorrhœa; and excessive smoking.

Symptoms.—In its early stages the growth may quite escape the patient's notice, and medical aid may not be sought until after ulceration, when pain sets in. Or the first sign that anything is wrong is the discovery by the patient of a hard lump in the neck behind the angle of the jaw, from secondary enlargement of the lymph nodes; so that, while it is a sound surgical rule to search for and *to find* the focus of infection in all cases of enlargement of the lymphatic glands, this rule is most imperative in the region under discussion, particularly in patients at or after middle life. The lymph nodes are early implicated in cancer of the pharynx, and may attain to a considerable size while the primary growth is still so small as to baffle discovery (Bland-Sutton).

The later symptoms associated with cancer of the throat are: shooting pains, increasing difficulty in opening the mouth and in swallowing, salivation, fœtor of breath, and gradual decline in the patient's powers until death occurs, in something under a year from the first discovery of the disease.

Epithelioma may start anywhere in the pharynx, but its favoured sites are the pillars of the fauces, the base of the tongue, the soft palate and the tonsils. In women a common situation is in the laryngo-pharynx. (" Post-cricoid cancer," see p. 66.)

The growth before ulceration sometimes appears as a pinkish infiltration of the mucosa, to which it imparts a glazed and polished surface broken up by transverse " hacks " or shallow fissures into rectilineal plaques or islets. The infiltration may spread over considerable areas of the mucous membrane before ulcerating, and at this stage it is difficult, without microscopic examination, to make a correct diagnosis of the disease. At other times, the growth in its early stages assumes a granular or warty appearance. Sooner or later, ulceration occurs, and presents the usual typical cancerous appearance, with elevated, indurated edges and rough, irregular granulations.

The hard infiltration of the edges is a diagnostic feature of much importance; thus **all suspicious pharyngeal ulcers should be palpated with the finger** (protected by a rubber finger-stall).

In the later stages, the epitheliomatous growth and ulceration spread all over the pharynx, until, it may be, tonsil, faucial pillars, uvula, and soft palate are occupied by a ragged, foul ulcer, with a hard base, and raised indurated edges.

The lymphatic glands at or a little below the angle of the jaw also enlarge rapidly, and may break down and ulcerate. Death results from inanition, exhaustion, septic pneumonia, or from hæmorrhage, which is a prominent feature of the final stages.

Diagnosis.—In the early stages it may be difficult to decide whether we have to deal with a cancerous or a syphilitic deposit. In these doubtful cases, where the appearances presented are so indefinite that a decision cannot be arrived at from clinical evidence alone, a piece of the diseased tissue must be removed and examined microscopically. The clinical facts which weigh most strongly in favour of cancer are—(1) the induration of the base and edges of the ulcer; (2) the early enlargement of the lymph nodes at the angle of the jaw.

We must never forget, however, that there may be a gummatous basis to the cancer. In other words, the lesion may be a mixed one. These are the cases which manifest much improvement when first treated with potassium iodide, but which betray their true character when, as time goes on, the preliminary improvement is not maintained.

Sarcoma.—The faucial and lingual tonsils share with the testicles the reputation of being a favourite site for lymphosarcoma—reputed to be the one form of sarcoma which tends to infect the neighbouring lymphatic glands. The other varieties of sarcomata also occur in the pharynx.

Sarcoma of the tonsils occurs as a smooth, ovoid, rapidly growing tumour, with a pallid, almost waxen appearance.

Its growth, extension, and fatal issue may be tragically rapid, and in such cases ulceration is unusual. On the other hand, many cases proceed much more slowly, so that sarcoma in this region is generally considered to be less dangerous than epithelioma. In the slower cases, ulceration and fungation are common.

The prognosis of sarcoma depends upon the type of the growth, and the effects of treatment. The lympho-sarcomata are the most malignant.

Treatment of Cancer of the Throat (Epithelioma).—(1) In the early stages before ulceration, or after ulceration while the disease is still limited in extent and in depth, and before any cervical gland enlargement can be felt, radical excision of the growth, either with the knife, or, what is better, with the blade-terminal of a diathermy apparatus, affords the patient his best chance.

When the disease is limited to a tonsil, that organ is removed by dissection as in the ordinary tonsillectomy operation.

At the same time, or if necessary, at a later *séance*, the gland-bearing connective tissue of the same side of neck with the cervical glands should be removed through an external incision.

(2) In a later stage, when the epithelioma has invaded the cervical glands, a similar but more extensive pharyngeal excision will be required, while the gland operation is performed as in the former case, care, however, being taken to dissect wide of the infected glands, so as to prevent sowing the open wound with cancer cells.

In the more extensive cases it is advisable to tie the external carotid artery as a preliminary step to the operation, whether the cold knife or the diathermy knife is to be employed.

(3) In still later stages, when the cervical infiltration has passed beyond the limits of the infected glands, even then it may be possible to prolong the patient's life for a year or more in usefulness and comfort by the frequent and judicious use of diathermy.

Diathermy is a modification of the high frequency current. In its passage through the tissues, this current develops heat, and if two terminals be brought into contact with the body, one of which covers a large superficial area of skin while the other is small, the heat concentrated around the latter may be made so great, by strengthening the current, as to char the tissues around it, while, at the same time, the zone surrounding the charred area undergoes heat coagulation. Thus, if applied to a cancerous ulcer or growth, in addition to the area visibly cauterized, there is set up in the deeper tissues a destroying action which results in a more extensive destruction of the tissue than is evident at the moment. (It should be noted that the terminal itself does not become heated. It is never red-hot as the electric cautery is.) The coagulation slough formed in this way separates in the week or ten days following the application. But its separation seems to be effected aseptically. There is little or no reaction, either local or general, and practically no pain.

Experience has proved diathermy to be of great value in pharyngeal cancer, particularly of the inoperable type. By direct and repeated applications in this latter variety it is possible to slow down the rate of growth and of extension, and to remove the pain and soreness, so that the patient can swallow, and can go on working and living his normal life for months longer than one might reasonably have expected. One cannot claim for it that it cures the disease.

In applying the diathermy, a general anæsthetic is required, and it is advisable not to prolong the exposure for more than twenty minutes, as it has been found that a reactionary pyrexia may follow too prolonged an exposure, and, indeed, may prove fatal.

Another point requiring caution is that diathermy is unsuitable for cancers growing close to the bone, as the bone is destroyed by the heat.

The limits of usefulness of diathermy are not yet settled, as it can be used for many other conditions besides pharyngeal cancer. I have employed it to remove tonsils and to eradicate synechiæ of the nose. And a useful suggestion of W. Stuart-Low, which I can support, is to reduce the size and rate of growth of inoperable cancerous lymphatic glands by multiple diathermy puncture after exposure of the gland by a skin incision, which is immediately closed.

Sarcomata of the pharynx may also be treated by excision with the knife or diathermy point. But perhaps the radium therapy is the most successful of all methods for this variety of malignant disease. A tube containing 50 to 100 mgm. of the radium salt, suitably screened, or else two or more fresh radium-emanation tubes are buried in the body of the sarcomatous tumour, and left there for a period of from eight to twenty-four hours, according to the size of the tumour. The results are sometimes dramatic in the rapid and complete disappearance of the tumour. Recurrence, however, is usual.

The influence of radium upon epitheliomata of the pharynx, on the other hand, seems to be less favourable than that of diathermy. Sometimes the radium seems actually to encourage the cancerous growth.

POST-CRICOID CANCER

(Cancer of the Laryngo-Pharynx)

Etiology.—The factors which lead to the selection of this site by the disease are unknown. It has been referred to insufficient mastication (Dundas Grant), or to the habitual drinking of hot iquids. It seems to be commoner in females than in males, and not infrequently appears in the third or fourth decade of life.

Symptoms.—Post-cricoid cancer is generally latent until it has become annular, or until it is so extensive as to interfere with the passage of food, and then the patient comes for relief of that symptom. The dysphagia is gradual in onset, but progressive. Hyper-secretion of saliva, which remains about the upper aperture of the hypopharynx and larynx, is visible on laryngoscopy. The disease, when fully developed, gives to the voice a characteristic muffled tone (W. M. Mollison). Pain is sometimes early, but more often late, and is most marked on swallowing. The progress of the patient downhill is rapid as in œsophageal cancer, and the end comes from inanition and general weakness, and may be precipitated by septic pneumonia if the disease extends upwards to involve the orifice of the larynx.

Diagnosis.—In some cases the laryngeal mirror shows the upper end of the growth beginning to involve the pharyngeal or posterior aspect of the laryngo-pharyngeal wall, as a reddish, irregular and nodular infiltration with some œdema above and around it. Occasionally the œdema is all that is visible. One or other vocal cord may be immobilized from interference with the recurrent laryngeal nerve.

In these cases hypopharyngoscopy will sometimes reveal the disease. (See p. 93.)

But as often as not there is no sign of any disease visible on indirect laryngoscopy, and only the direct examination can be relied upon to exclude the disease or to disclose it. On using this method, the cancerous growth appears as an area of infiltration and ulceration, readily bleeding, and interfering with the free downward passage of the tube about the level of the cricoid (lower pharyngeal isthmus). For this reason a tube of moderately small calibre only should be used when cancer is suspected, and no attempt should ever be made to push it on into the œsophagus beyond the growth, as the cancerous wall is extremely friable, and its rupture would be followed by rapidly fatal mediastinitis (see p. 195.) A piece of the growth may be removed for microscopic examination.

Treatment.—(*a*) *Radium* has many advocates, and it is, indeed, sometimes very useful, especially in inoperable cases. A radium (or radium-emanation) tube of 50 to 100 mgm., suitably screened and secured by a thread, is inserted by means of suitable forceps through the direct tube, and allowed to remain *in situ* for from eight to twelve hours, according to the size of the growth. The patient meantime lies quietly in bed, and is kept under the influence of morphia; no food or liquid is allowed, thirst being controlled by rectal salines.

The exposure is followed by a well-marked reaction, as is evident in the increased swelling and œdema of the parts in the neighbourhood of the growth. This slowly subsides in the course of about six weeks, and with its disappearance the growth undergoes a rapid atrophy until, in favourable cases, no trace of the original disease can be found. Even scarring may not be perceptible.

After from three to six months of freedom from dysphagia and pain, recurrence usually sets in, either locally, in which case the throat symptoms reappear, or in the glands, in which case the disease may go on to its termination without giving rise to much pain or distress.

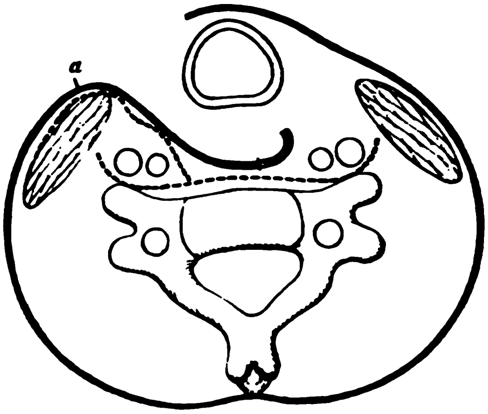


FIG. 14.—Operation for removal of post-cricoid carcinoma. Restoration of lumen of hypopharynx. (From W. M. Mollison.)

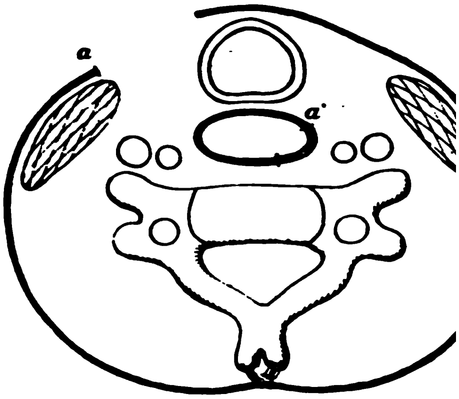


FIG. 15.—Restoration of lumen complet

In a few cases, the application of the radium seems to do harm. The growth is stimulated, the pain becomes more severe, and the patient rapidly loses ground.

(*b*) *Diathermy* is unsuited for cancer in this situation.

(*c*) *Operative Treatment* may be tried when the growth does not entirely surround the pharyngeal opening; if, that is to say, there is a strip of sound mucous membrane that may be left to carry on the œsophageal connection.

Two methods have been tried. First, excision of the larynx, and then removal of the cancer-bearing mucosa behind it, the sound strip referred to above being left. A feeding tube is inserted from the nose, and left in the gullet for at least three weeks. The wounds made are stitched up entirely. (E. Waggett.) (See p. 141, Laryngectomy.)

Or secondly, an incision is made to one or other side of the middle line from the level of the hyoid bone to near the suprasternal notch. From the upper end of this incision, another is made along the hyoid bone to the anterior border of the sternomastoid. The triangular area of skin so defined is dissected with its platysma from the deep fascia, thus exposing the carotid triangle. The greater hyoid cornu and the thyroid ala are freed and removed, care being taken to avoid the superior laryngeal nerve. The pharynx is opened by a vertical incision, the growth seen, defined and removed by stripping it from the cricoid and cutting round it, the healthy strip being left to unite pharynx above with œsophagus below. A tube is passed from the mouth to the œsophagus for feeding, and the neck wound is sutured, but provision should be made for free drainage.

The lumen of the new tube may be made up by a flap of skin turned in from the neck. (Wilfred Trotter.) (See Figs. 14 and 15.)

For *Malignant Disease of the Naso-pharynx,* see p. 390.

SYPHILIS OF THE PHARYNX

Hereditary Syphilis in infancy sometimes manifests itself in the tongue and pharynx in flattish condylomata and mucous patches, or in shallow ulcers on the fauces and tonsils, but these appearances are not common.

About puberty gummatous perforations of the palate and deep ulcerations of the pharynx are sometimes seen.

Acquired Syphilis : The Primary Sore.—When an individual is infected with syphilis through the mouth, the most usual site for the primary sore to form is on one of the tonsils, and as the local reaction may be slight, and the patient be unaware of its presence, this is one of the varieties of primary sore which often give no hint of their existence, and leave no trace behind them.

The primary sore may also be seen on the lips, tongue, and faucial pillar.

Cause.—The conveyance of the infection to the mouth or pharynx may be due to kissing a person with primary or secondary syphilis of the pharynx or mouth ; to infection from drinking vessels ; as well as to unnatural practices.

Symptoms.—The patient experiences a sore throat, the pain and discomfort of which are one-sided. It lasts for from four to six weeks, and is accompanied with considerable enlargement of the glands at the angle of the jaw on the same side.

Septic infection of the locality may transform the appearance of the case into one resembling acute septic pharyngitis with high temperature and constitutional disturbance.

Diagnosis.—Unless the possibility of syphilis occurs to the physician, the true nature of the case is very apt to be overlooked. Chancre of the tonsil manifests no very striking or characteristic phenomena. The tonsil is inflamed, its size is greater than normal, and it may present an erosion or an ulcer with or without raised edges. The appearance may thus simulate Vincent's Angina or epithelioma. If suspicion is aroused by the chronicity and by the enlargement of the glands, the diagnosis can be thereupon established by the discovery of the spirochæta pallida on needling the tissues deep to the ulcer; by the Wassermann reaction becoming positive; or by the development of secondary symptoms.

The disease should not be diagnosed as syphilis solely from the clinical appearances in the throat.

Treatment.—The constitutional treatment of syphilis by one of the salvarsan group and by mercury should at once be instituted.

Locally, Sir St. Clair Thomson advises the careful application of pure carbolic acid to the dried surface of the ulcer, and septic infection should be prevented by the use of an antiseptic mouthwash and gargle.

Secondary Syphilis of the Pharynx.—Syphilis in the secondary stage affects the pharynx as part of the general constitutional reaction and appears about the same time as the secondary skin rash; that is from five to twelve weeks after infection, and sometimes later. This is the most infectious stage of the disease, and the lips, tongue, mouth and their secretions are all virulent.

Symptoms.—There is sore throat affecting both sides of the pharynx, and lasting from three to six weeks, with moderate glandular enlargement. At the same time, the constitutional phenomena of syphilis are manifesting themselves in the syphilitic roseola; the evening temperatures; the headaches; the gland enlargement and induration; and the loss of hair. But all of these symptoms are often so mild as to attract no attention.

Signs.—The throat shows general congestion, symmetrical and sharply defined at the upper limit of the soft palate. Mucous patches—slightly elevated bluish-white areas, with a filmy adherent surface, the so-called "snail-tracks," are common, being symmetrically disposed on the anterior aspect of the faucial pillars where the dorsum and edges of the tongue come into contact with those parts when the mouth is closed. They are also found on the sides of the tongue, and on the surface of the tonsils. At a later stage, symmetrical shallow ulceration is occasionally seen, which, if neglected, may manifest an excavating tendency until its appearance comes to resemble that of the deeper tertiary ulcer.

Diagnosis.—A sore-throat in an adult which lasts any longer than three weeks should always raise the question of syphilis.

The "secondary throat" is distinguished from a simple pharyngitis by its chronicity when untreated, and by the mucous patches, which are unmistakable when once seen. The inspection should be made by daylight or other white light.

Prognosis.—Secondary syphilitic pharyngitis passes off spontaneously in most cases in four to six weeks, but if untreated, as already noted, deep ulceration may supervene. Under treatment, the disappearance of the throat phenomena is rapid.

Treatment—General.—The modern constitutional treatment of syphilis consists in the administration of one of the salvarsan group of drugs generally intravenously, together with the regular administration of mercury by the mouth, by inunction, or by intramuscular injection, assisted by iodides when there is much infiltration or ulceration.

Neo-salvarsan or novarsenobillon (0.75 of a gram) is dissolved at the moment of use in a fixed quantity of distilled water. One of the veins on the front of the elbow is usually selected for the intravenous injection of the solution. Its exposure by dissection is unnecessary, as with a suitable needle the lumen may be safely reached through the skin. It is well to inject a little saline first to make sure that the needle is accurately within the vein, otherwise the arsenical solution may find its way into the tissues, and set up sloughing.

The injection may be repeated every month or so for three or four months, while, in the interval, mercury is being administered.

The salvarsan drugs are specially valuable in laryngeal syphilis where iodides are inadmissible, and a prompt effect is required to relieve stridor and dyspnœa. (See p. 115.)

Contra-Indications.—They should not be employed when there is any nerve deafness or vertigo, as their immediate effect upon syphilitic lesions is to set up a congestion in the neighbour-blood (the Jarisch-Herxheimer reaction), and this may permanently destroy the functions of the acoustic and vestibular nerves. They are also contra-indicated if there is albuminuria.

Mercurial treatment:

(1) By the mouth.

℞ P : Hydrarg : c̄. Cret : gr. 1
P : Ipecac : Co : gr. i
in pill, —M

is given three times a day for three months at a time with a month's interval between the courses. The pills are continued for upwards of two years.

(2) By inunction.

℞ Ungt. Hydrarg. gr. 40
Adip. Lan. gr. 20 —M

is rubbed for half-an-hour daily into the inner side of the thighs, the flexor aspect of the arm, and the abdomen, alternately.

This is carried on with a day's rest once a week for (1) six weeks, followed by a three months' interval; and (2) a second course of six weeks, followed by another three months' interval; and (3) a third course of one month followed by an interval of six months; and (4) a fourth course of one month followed by an interval of six months; and (5) a final course of three weeks.

(3) Intramuscular Injections.—Lambkin's Grey Oil containing 1 gr. of mercury in 10 minims, is recommended.

The oil is liquefied by warming and stirring, and the syringe, which is furnished with a wide-bore needle, aspirates 10 minims. The needle is plunged vertically into the fleshy buttocks, and after insertion, the barrel is detached from the needle to make certain that no vein has been punctured. If blood comes from the needle it should be withdrawn and inserted at another place.

Of the above methods, the oral is the least troublesome, but it is also the least effective. The intramuscular method is the most certain and convenient, but it is not quite free from risk. Inunction is an unpopular method in ordinary life.

Iodides are used in the tertiary stages when there are gummata and ulcers. Iodide of potassium alone is selected, save when large doses are given, in which case the iodides of sodium and ammonium may be combined with it. The drug is given by the mouth half an hour before food, beginning with from 6 to 10 grains, and increasing until its effect becomes visible. As much as 180 grains, or even more, may be given every twenty-four hours.

In severe cases of syphilitic destruction of the nose, throat, or larynx, the patient should be kept in bed and well supplied with nourishing food, as the disease in these conditions is not only frequently obstinate, but it is also occasionally dangerous to life. (See also p. 289.)

Locally.—A gargle and mouth wash of

℞	Potass. Chlor.	grs. 10
	Lot. Nig.	℥ i
	Aq.	℥ i —M

is used after each meal.

For the mucous patch, the application of chromic acid (grs. x to ℥ i) or silver nitrate (grs. xx to ℥ i) is useful.

Tertiary Syphilis of the Pharynx.—Three varieties of lesion are met with: ulceration, the gumma, and diffuse infiltration.

With the exception of the ulcer which sometimes supervenes upon the secondary syphilitic pharyngitis, the tertiary lesions appear late in the history of the disease, namely, from the eighth to the fourteenth year after infection.

The *gumma* generally attacks the soft and hard palate near the middle line, but it is seen also on the tonsil, on the posterior pharyngeal or naso-pharyngeal wall, and on the base of the tongue. It seldom comes to our notice until it has begun to ulcerate, and then there is a bright red and swollen area of infiltration surrounding a deep ulcer with punched-out edges, and a yellow, tough, wash-leather base. In the soft palate, the deepening ulceration, which often commences on the naso-pharyngeal aspect of the palate, has generally advanced to perforation before the surgeon sees the case. When the hard palate is affected, the bone becomes involved in the disease, and perforation leads to a communication being formed between mouth and nose. Neglected, such infiltration and ulceration may extend until there is widespread destruction of tissue in the pharynx and palate.

Symptoms.—The subjective phenomena are often unobtrusive, and the amount of pain experienced trifling. Nasal phonation or the passage of liquids into the nose on swallowing may be the first symptoms to attract the patient's serious attention.

Tertiary ulcers are readily seen on inspecting the throat, but it sometimes happens that when situated on the posterior aspects of the palate or tonsils they escape discovery. Any localized congestion of these regions should, therefore, always lead to an inspection of their hidden posterior surfaces with the post-rhinoscopic mirror, or with the naso-pharyngoscope.

Diagnosis.—Chronic pharyngeal ulceration is, to mention the commoner types only, either syphilitic, tuberculous, lupoid, or cancerous in nature, and of these the syphilitic is by far the most common.

The *syphilitic ulcer* is relatively painless, of sudden origin and rapid course; it presents a purplish edge, deep punched-out sides, wash-leather base, and it is not noticeably hard or indurated. It is associated with the other signs of syphilis —the Wassermann reaction is usually positive, and the disease shows rapid improvement under anti-syphilitic treatment.

The *tuberculous ulcer* (see p. 76) is exceedingly painful, of insidious origin, and slow course; its edges are smooth, and the mucosa around is pallid; the sides of the sore are very shelving, the base is grey and pale, and there is no induration. There are no syphilitic stigmata except in mixed cases, which, however, are not infrequently encountered. The effect of anti-syphilitic remedies, however, is negligible, even in mixed cases.

The *lupus* ulcer is painless; its surface is grey, dry, and granular; its floor, and the mucosa around it are infiltrated with lupus nodules, and deposits are found also elsewhere, usually in the nose.

The *cancerous ulcer* is painful, with thickened, irregular and hard edges, and with hard, infiltrated base. The secretion is foul, and, if advanced, there is interference with movement. The cervical glands are enlarged and hard

For diagnosis from Vincent's Angina, see p. 36.

In any doubtful case, a portion of the ulcer, including the margin, should be punched out and examined microscopically.

The *Prognosis of Tertiary Syphilis of the Pharynx* is most favourable when the disease is in its earliest stages, because then the loss of tissue is but trifling, and the amount and degree of cicatrization is limited. More advanced cases are favourable in so far as the cure of the active disease is concerned, but loss of tissue cannot be made good, perforations through the palate remain open, and the cicatrization which follows the healing of extensive and deep ulceration often leads to serious deformity, and in later years may form the starting point of epithelioma.

Cicatrization leading to occlusion of the naso-pharynx has already been dealt with (p. 24).

Treatment.—General treatment is in most cases all that is required, the ulcers healing up rapidly. (See p. 71.) Locally, if foul, ulcers may be cleansed by regular spraying with fresh hydrogen peroxide solution (5 to 10 vols. per cent.).

Persistent perforations of the palate can be blocked with suitable obturators.

TUBERCULOSIS OF THE PHARYNX AND TONSILS

Excluding lupus, there are two varieties of tuberculosis of the pharynx.

The First and Commoner Variety affects the tonsils, and is due to their invasion by the bovine tubercle bacillus. As it is a milk infection, it is commonest in childhood. In this type the disease occurs in the substance of the tonsil; it does not produce ulceration; it is not, at this stage at all events, accompanied with pulmonary tuberculosis; but it is associated with tuberculosis of the cervical glands, of which, indeed, it is the precursor and introducer. (A. P. Mitchell.)

Symptoms.—There are no symptoms referable to the pharynx. The tonsils are generally shrunken and atrophied, although the cervical glands are enlarged.

Prognosis.—Locally, the disease in the tonsil runs a quiet and uneventful course, and its only importance—but this is great—is that as long as it remains in existence, the disease in the cervical glands is incurable.

Treatment.—In all cases of tuberculosis of the cervical glands, whatever treatment be adopted for them, **the tonsils must be dissected out, whether they appear to be enlarged or not**, in order to seal up the channel by which the tubercle bacillus attains entrance.

At the same time no milk is to be consumed by the patient but what is sterilized by heat.

The second and rarer variety is the surface ulceration of the pharyngeal mucosa found occasionally in phthisis pulmonalis, of which it is the sequel, the infective sputum being responsible for the disease. It is found in adults, and is not associated with tuberculosis of the cervical glands. The tuberculous pharyngeal ulcer is usually a late phenomenon in the course of pulmonary disease, but occasionally it appears early and spreads to involve extensive superficial areas of the pharynx.

Symptoms.—Tuberculous ulceration of the pharynx gives rise to pain on swallowing so severe that the patient prefers to go without food sooner than endure the distress that swallowing produces.

On examination, the ulcer appears as a more or less extensive, shallow grey area, with eroded, nibbled edges. Its favourite sites are the base of the tongue, the posterior wall, and the uvula and soft palate.

The *prognosis* of this variety of tuberculous ulceration of the pharynx is bad. By interfering with nutrition, it hastens the downward course of the pulmonary disease.

Treatment can only be palliative. It may be possible to reduce the sensitiveness of the ulcer sufficient to enable the patient to swallow by insufflating upon it the powder of orthoform, or by spraying it with sol. cocaine (5 to 10 per cent.). Morphia hypodermically should be employed if the pain is severe.

Acute Miliary Tuberculosis of the pharynx is described. It appears as minute discrete tubercular deposits visible on inspection, and occurs in the terminal stages of phthisis pulmonalis. It is very rare.

(For tuberculosis of the retro-pharyngeal region, see p. 46.)

LUPUS OF THE PHARYNX

Lupus of the pharynx is almost invariably secondary to lupus of the nose or face. As is the case when the disease attacks the integument, lupus of the throat is insidious in onset and very chronic in its course.

Subjectively, only slight soreness or some cough or dysphagia may be complained of. Pain is usually absent.

Physical Signs.—There is a slow deposit of lupus nodules, first affecting the uvula or pillars of the fauces, and presenting the appearance of smooth, hard, rose-coloured nodules embedded in the mucous membrane. Their pinkish, apple-jelly-like colour is well brought out by adrenalin. When widely diffused, they distort the shape of the parts, thickening fine folds, blurring clear edges, and imparting also a certain stiffness or wooden appearance to structures normally flaccid and movable. Later on, serpiginous, "worm-eaten" *ulcers* appear, with velvety, indolent bases. As in the skin, Nature's attempts at healing are marked by the presence of cicatrices.

Prognosis.—Lupus of the pharynx is amenable to treatment. The danger is that the disease may extend to the larynx (see p. 125), and eventually lead to pulmonary tuberculosis.

Treatment—General.—Generous feeding with malt, cod liver oil, cream, and tonics, arsenic especially, are indicated. *Locally*, each nodule is punctured with the galvano-cautery, or diathermy needle. A number of applications may be required before the whole of the disease areas have been overtaken.

Pharyngomycosis, or to speak more accurately, **Keratosis Pharyngis**, is the growth of the leptothrix fungus in spots where the epithelium of the pharynx has undergone cornification (keratosis).

Symptoms.—The disease is generally discovered accidentally, as in most cases it induces no subjective phenomena. Sometimes, however, patients complain of irritation or pricking, for which no other cause than the keratosis can be found.

On *inspection* the faucial, and sometimes the lingual, and the naso-pharyngeal tonsils are seen to be studded with small, white spots, which, on being grasped by forceps, are found to be tough and horny outgrowths from the mucous membrane, very difficult to detach from their position.

Their irregular distribution and disposition irresistibly remind one of gravestones in a country churchyard. Fortunately, there is nothing so gloomy in their significance. The disease is insidious and persistent, but apparently of no importance. Treatment has no effect upon it ; even the removal of the tonsils is only followed by the re-appearance of the keratosis in the tonsillar fossa.

Its only interest lies in the diagnosis. It is distinguished from lacunar tonsillitis by chronicity and apyrexia, and by the fact that its spots consist of tough projections ; and from diphtheria by the absence of any coalescence or confluence of the disease-spots.

Keratosis has been seen in the larynx giving rise to hoarseness and pain.

NEUROSES OF THE PHARYNX

Sensory.—Anæsthesia of the Pharynx is—

1. *Bilateral*, in diphtheritic paralysis, in hysteria, bulbar

paralysis, and insanity. Hysterical anæsthesia is the most common form.

2. *Unilateral,* from hysteria, or from interference with the glosso-pharyngeal nerve by tumour, etc., in which case it is accompanied by symptoms due to loss of function in the parts supplied by this nerve.

Hyperæsthesia and Paræsthesia of the pharynx are very common. The patients are generally sufferers from hysteria or neurasthenia. The **globus hystericus** is the most typical hysterical paræsthetic condition, but all kinds of feeling may be referred to the throat by hysterical or nervous people.

Diagnosis.—Every care must be taken to exclude organic disease in the supposed functional neuroses of the pharynx. Incipient phthisis or malignant disease may be responsible for the perverted sensation, and all patients with such a symptom must be examined so as to exclude these diseases. It is a wise rule always to make an œsophagoscopic examination in women with paræsthesia pharyngis.

Further, in a certain number of cases the sensation seems to be due to enlargement of the lingual tonsil. At all events, it is not infrequently removed or relieved by the application of the cautery to this area. (See p. 606.)

Treatment.—The anæsthesia of diphtheritic paralysis is treated according to directions already given (p. 41).

Anæsthesia from bulbar paralysis, being combined with motor paralysis, is associated with imperfect deglutition and the food is apt to enter the air-passages and to set up pneumonia. In this condition the patient must be habitually fed through the stomach-tube.

For the *paræsthesia* of nervous people general treatment alone should be adopted. Operative procedures on the pharynx are harmful as a rule, save in the reflex conditions which are clearly due to hypertrophy of the lingual tonsil, in which case that disease should be treated. (See p. 606.)

If a patient is convinced of the presence of a fish-bone or other foreign body in the pharynx, it is legitimate, sometimes, to provide such a body for his satisfaction. Cure may follow.

If, after thorough examination repeatedly carried out, no organic basis is found for the sensation, the patient should be told that his disease is neither cancer nor tuberculosis, as the fear of these diseases is often responsible for the paræsthesia.

Motor Paralysis of the soft palate is most commonly due to diphtheria. It may also be produced by some interference with the nuclei of the vagus and spinal accessory nerves in the medulla, as in tabes, bulbar paralysis, tumours and apoplexy of the medulla, and syringo-bulbia; or by a lesion, such as a gumma, syphilitic meningitis, or a neoplasm, commanding the nerves at the base of the brain, in the jugular foramen, or in the neck. A paralysis which is myopathic arises from inflammatory lesions of the pharynx; probably also, the temporary paralysis of the soft palate that occasionally follows tonsillectomy is also myopathic in origin. (See p. 62.)

Functional paralysis of the soft palate is occasionally seen, but it is very rare.

The belief that peripheral facial paralysis produces paralysis of the soft palate is no longer credited.

Symptoms.—The patient speaks through his nose (rhinolalia aperta), and solids are more easily swallowed than liquids, the latter returning through the nose. The patient cannot forcibly expel his breath from the lips as in whistling, and if a smoker, has difficulty in " drawing " his pipe.

The interference with the deglutition may cause frequent choking attacks from liquids finding their way into the larynx.

On *examination* in *bilateral* paralysis, the soft palate is seen to be immobile, remaining flaccid when the patient phonates ' Ah," and when the back of the pharynx is touched.

In *unilateral* paralysis attempted movement drags the paralysed side and with it the uvula towards the sound side.

Prognosis depends upon the cause. The diphtheritic and inflammatory types get well. Bulbar pharyngeal paralysis, on the other hand, is of evil import, as it marks the terminal stage of that affection.

The *Treatment* of pharyngeal paralysis is applied to the cause.

Spasmodic Neuroses.—Pharyngeal *tonic spasm* occurs in tetanus and hydrophobia. Otherwise it is functional.

Clonic spasm of the palate and pharyngeal wall with contractions, repeating themselves from 60 to 160 times a minute, is sometimes seen. It has been reported as occurring in softening of the brain. Treatment is of no avail.

For *Diseases of the Lingual Tonsil, and Base of the Tongue* see p. 605.

CHAPTER IV

EXAMINATION OF THE LARYNX AND LARYNGEAL TECHNIQUE

LARYNGOSCOPY

There are three methods of inspecting the larynx—one indirect with the laryngeal mirror, and two direct, namely: with the tube-spatula; and by suspension. As the indirect is the most commonly employed, we shall describe it first and most fully.

Indirect Laryngoscopy.—Use the largest laryngeal mirror the patient will tolerate (Fig. 16). Sitting opposite to him, ask the patient, who is to be seated well back into his chair, with the upper part of his body leaning slightly forward, to open the mouth and put out the tongue. Wrap one or two folds of fine cotton or linen round the point of the tongue, and hold it between the finger and thumb of the right hand, resting the other fingers of that hand on the patient's chin. Do not pull on the tongue, nor hold it so far down that it rubs on the lower incisor teeth. Direct the rays of the light from your forehead-mirror into the patient's mouth; then, holding the laryngeal mirror in the left hand, warm it by plunging it in hot water, or by holding it face downward over a spirit lamp or Bunsen gas flame. Test the heat of the mirror by placing its back against the back of the hand holding the tongue. Ask the patient to breathe through the mouth, and to keep his eyes fixed on your face. Insert the warm mirror, face downward, into the mouth without letting it come into contact with the tongue; carry it back

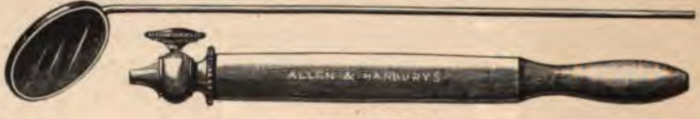


FIG. 16.—Laryngeal Mirror.

until it reaches the base of the uvula. Keeping the mirror in the middle line, and while the patient is drawing deep, easy breaths, push the soft palate upwards and backwards, until it almost touches the posterior pharyngeal wall. Then, without attempting to observe anything, withdraw the mirror, in order to give the patient a rest, and after a few moments, having again warmed it, reinsert it as before. The image in the mirror, while it is in the position just indicated, will be the base of the tongue and the tip of the

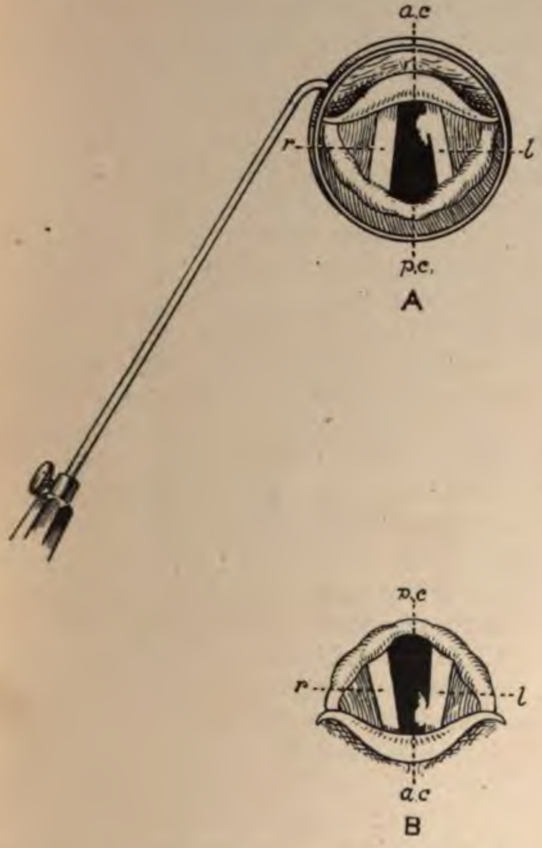


FIG. 17.—Indirect Laryngoscopy (from Morell Mackenzie's "Diseases of the Throat and Nose").

A, the image reflected in the mirror of *B*, the larynx.
a.c. the anterior commissure.
p.c. the posterior commissure.
r. the right vocal cord.
l. the left vocal cord, upon which a neoplasm is figured.

epiglottis. Raise the handle so as to lower the mirror somewhat in the pharynx, alter the direction of the plane of the reflecting surface to a more open angle, at the same time carry the handle to the side of the mouth, and you will obtain, in most cases, a full view of the interior of the larynx. At this moment it is advisable to ask the patient to phonate (to sing " E-e-e ")* in order to distract his attention at a critical moment, and to enable you to find your landmarks by catching sight of the vocal cords as they come together. The patient should be directed to alternate phonation and inspiration for a few times. By doing so the movement of the cords are most plainly made visible. Remember that you are looking at an inverted image, and that the right side of the patient is on your left. (Fig. 17.)

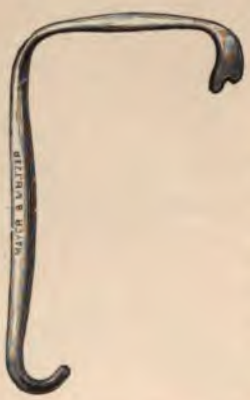


FIG. 18.—Escat's Epiglottis Retractor.

The difficulties one encounters in practising laryngoscopy are nearly all surmounted as one gains command over the instrument. A calm and confident bearing, free from fussiness and irritability, is the best method of reassuring a nervous patient. It frequently happens, however, that hypersensitiveness of the fauces, and a tendency to gagging and retching render the examination impossible, even in the case of a calm patient. This reflex irritability may be overcome by spraying a 10 per cent. solution of cocaine over the fauces and base of the tongue. Again, a pendant epiglottis, lying over the larynx like a half-closed lid, a not infrequent condition, may conceal the interior. This difficulty may be avoided by placing the mirror as far down and back in the pharynx as possible. If this manœuvre fails, one may employ an epiglottis retractor, the ends of which are placed in the valleculæ, and, when pulled upon, drag up the epiglottis. A convenient method of retaining the epiglottis in this position for operating purposes is to pass a stitch through it by means of Horsford's epiglottis needle (Fig. 24), and have it held by forceps. Again, many people when sounding " Eh-h-h " cannot do so without arching the tongue, and this, of course, shuts off all view of the mirror. If you cannot teach the patient to keep his tongue flat when phonating, ask him to hold his tongue himself, or get an assistant to do so, while with the hand thus disengaged you hold down the tongue by means of a tongue-depressor.

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* The patient under these conditions cannot say or sing "E-e-e," but in his effort to do so he says "Eh," and that is the sound we wish to obtain. Do not let him say "Ah."

Laryngoscopy should be practised on healthy people, not only because it is necessary to become perfectly familiar with the normal appearances, but also because when the larynx is diseased, examination with the laryngeal mirror is almost always more difficult than in health.

People not infrequently come before us in whom the laryngeal reflex is so sensitive that examination with the laryngeal mirror is impossible, even after using cocaine. In such cases Potass. Brom. in gr. x doses three times a day for a few days will reduce the irritability to a manageable degree. On the other hand, in cases that have to submit to frequent examination extraordinary tolerance of the mirror is soon acquired. This applies even to children. In infancy, of course, it is impossible to practise indirect laryngoscopy, but at this time of life the direct or the suspension method may be used.

The Normal Image.—(Fig. 19.)

After studying the base of the tongue, with its lingual tonsil and veins, bounded in front by the V of the circumvallate papillæ and the foramen cæcum, note the central glosso-epiglottic ligament, or frænum of the epiglottis, the right and left valleculæ, or glosso-epiglottic fossæ, and the right and left lateral glosso-epiglottic ligaments, bounding these fossæ laterally. Next, direct your attention to the epiglottis, which is the first feature to attract the eye in laryngoscopy. The epiglottis varies much in position and shape in different individuals, but one can almost always see both the anterior and posterior (or buccal and laryngeal) surfaces. Next, the pearly white or pale pink vocal cords readily catch the eye. During quiet breathing, they appear some little distance below the epiglottis, diverging as they pass backwards to their attachments to the arytenoid cartilages. They form thus a V, the anterior angle of which is known as the anterior commissure.

Towards the upper part of the image, note the arytenoid eminences, marking the small cartilages of Santorini which surmount the arytenoid cartilages proper. From the arytenoid eminence on either side to the epiglottis runs the ary-epiglottic fold, in which may be seen the elevation caused by the cartilage of Wrisberg. The ary-epiglottic folds are continued posteriorly into the mesial interarytenoid membrane. While in the position of rest, this membrane, which encloses the interarytenoid muscle, is taut; during phonation the approximation of the arytenoid cartilages by the contraction of the interarytenoid muscle throws it into folds.

Clinically, the larynx is divided into three portions :—

1. The supraglottic space, above the ventricular bands;
2. The infraglottic space, below the vocal cords, to the lower border of the cricoid cartilage; and
3. The glottis, which comprises the vocal cords and the ventricular bands, including the ventricles.

The space between the vocal cords is the chink of the glottis (rima glottidis), ever changing in shape and size with the varying positions of the cords. Study the position of the vocal cords during quiet breathing (abduction), deep inspiration (full abduction), and phonation (adduction). In what is known as the "cadaveric position," the cords occupy a position a little internal to that which they occupy during quiet breathing. In phonation the cords are approximated (adducted) until the space between them is represented by a single line. During quiet respiration, they lie between adduction and full abduction, approaching to the position of full abduction, which is assumed when the patient draws a deep breath. In appearance the cords are flat, with smooth surfaces and clean, even edges. External and parallel to the true vocal cords, and sloping upwards so as to form the lateral walls of the glottis, are the ventricular bands, or false vocal cords. Lying between the ventricular bands and the inner surfaces of the alæ of the thyroid cartilages is a small space, the sacculus laryngis, or ventricle of Morgagni, the opening of which may be seen between the vocal cords and the ventricular bands. One or more of the rings of the trachea can frequently

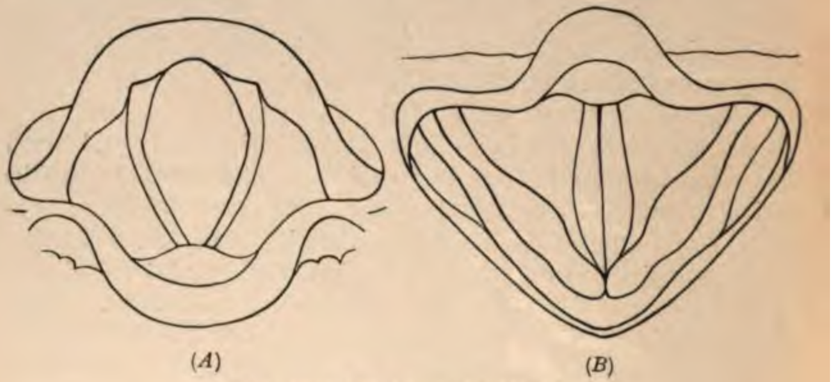

FIG. 19.—The Laryngeal Outline.

A. During Respiration (Abduction). *B*. During Phonation (Adduction).

be seen through the chink of the glottis, and very occasionally the division of the trachea into right and left bronchus is visible.

Observe the colour of the different constituents of the larynx: the epiglottis is yellowish, the vocal cords pearly white or pale pink, while the rest of the larynx varies from red to pink.

Abnormalities in Colour.—*Pallor* of the laryngeal mucous membrane will be found if the patient is suffering from anæmia. A definitely localized anæmia of the larynx, while the patient seems otherwise well coloured, raises the suspicion of phthisis.

Hyperæmia.—Apart from diseased conditions, nearly all of which induce a deepening of the colour of the larynx and a reddening of the cords, hyperæmia may arise from such simple causes as prolonged phonation, excessive coughing, and the inhalation of irritating substances.

Next observe in detail the **Epiglottis.** The epiglottis may be partly or wholly absent, generally as a result of old syphilitic ulceration.

Ulcers on the epiglottis may be syphilitic, tuberculous, lupoid, or cancerous. In the early stages of syphilis, mucous erosions of the epiglottis may appear, accompanying mucous patches in the mouth, fauces, and pharynx. Gummata breaking down form large, deep ulcers, generally on the tip or upper edge of the epiglottis. These ulcers may cause pain on swallowing, but the pain is slight in comparison with their size. They are usually large, irregular in outline, and solitary, and are surrounded by an areola of hyperæmia.

Tuberculous ulcers, formed by the disintegration of pale, smooth tubercular swellings, are, in the early stages at least, multiple. They are small, shallow, and circular, and occur chiefly on the laryngeal surface or upper edge of the epiglottis. The surrounding mucous membrane is pale. Pain is a marked feature. Later on, these small ulcers coalesce, and, becoming deeper, expose the underlying cartilage, which undergoes necrosis.

The lupous ulcer is preceded by the deposit of lupous nodules.

Epitheliomatous ulcers on the epiglottis are uncommon, save when the organ is attacked by extension from neighbouring parts. Cysts are not uncommon. Papillomata and other neoplasms are rare.

Inspect the **aryepiglottic folds** for mucous patches, gummata, and syphilitic ulcers, for tuberculomata and tuberculous ulcers. The movements of the folds may be impaired by œdema, or by chronic inflammatory thickening in chronic laryngitis, tubercle, lupus, or syphilis. The anterior aspect of the **interarytenoid region** is a favourite site for a tuberculous deposit. Indeed, the first sign of laryngeal tuberculosis is often to be found here. It appears as a slight elevation of the mucous membrane of a somewhat pale tint; this, after a time, breaks down and forms an ulcer, which frequently presents exuberant granulations (see p. 118). A pale œdematous swelling or fullness of the arytenoid eminences is also an appearance highly suggestive of tuberculosis. The interarytenoid region is a favourite site also for the growth of these warty excrescences which characterize the pachydermatous variety of chronic laryngitis, and it may therefore be difficult to decide whether the condition is pachydermia or tubercle.

When the view of the cords themselves is obscured or obstructed, information as to their mobility may be obtained by observing the movements of the arytenoid cartilages, which approach each other and recede with the adduction and abduction of the cords.

The **ventricular bands** may be swollen and red from inflammatory conditions. Their inner edge provides a favourite site for superficial ulcers in the secondary stage of syphilis. The ventricular bands take part, of course, in general laryngeal œdema, but if œdema is limited to them, the cause is probably tuberculosis, which frequently affects these parts or the enclosed ventricles. The ventricular bands may be the seat of tumours—polypi, papillomata, fibromata, and not infrequently epithelioma begins in these structures.

The **Vocal Cords** are examined—(1) as to their colour and shape; (2) as to their position at rest and when moving. Note any departure from the normal in either respect.

Note if the cords are inflamed or thickened, if the edges are rough, or seem ulcerated. Observe particularly the existence of warts or growths, and whether there is in such cases any impairment in the free movement of the cords. The neighbourhood of the vocal processes is frequently the site of small growths or protuberances. If the movement is impaired, notice whether one or both cords are affected. What position—adduction, the cadaveric position, abduction, or full abduction—does the affected cord take when at rest? If the true cords do not meet on attempted phonation, do the ventricular bands come together in a vicarious attempt to produce sound? Do the cords, during phonation, come into contact along the whole or only part of their free edge?

After the examination of the cords is completed, make the patient take some deep breaths, and direct your attention to the subglottic and tracheal regions. Subglottic tumours and deviations and stenosis of the trachea can frequently be seen in laryngoscopy.

Direct Laryngoscopy is the examination of the larynx by direct vision unaided by a mirror in the pharynx. There are two methods of direct examination; first, that by means of the laryngeal tube-spatula of Brünings or Chevalier Jackson; and secondly, that by means of suspension laryngoscopy.

Direct Laryngoscopy by means of the Tube-Spatula.—There are two types of spatula in use: Brüning's, which carries the illuminating lamp in the handle, and Mosher's, which carries its lamp at the distal end of the tube. (Fig. 20.) Both are introduced and used in the same way.

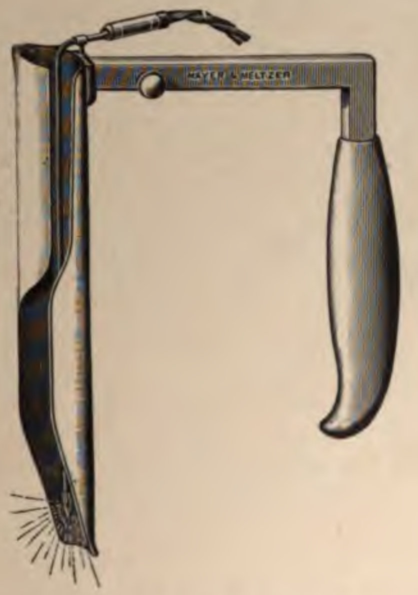

FIG. 20.—Laryngeal Tube-Spatula (Hill's modification of Chevalier Jackson's).

Method.—In adults the examination is made under local, in children, under general anæsthesia. Under local anæsthesia the patient is seated, either in a very low chair, or in what is known as Mouret's position; that is astride a chair and facing the back of it, with the body bent forward at the pelvis. Under general anæsthesia the patient is laid on his back, with the head on a level with the body, or slightly raised, and turned towards the operator, who stands on the right side of the table, or the head is fully extended on the table.

The tube having been warmed and lubricated, it is passed over the tongue either in the middle line, or what is often more suitable, from one or other angle of the mouth, under the direct vision of the operator looking into the tube. The first object seen is uvula, and whenever it becomes visible, the handle of the tube should be raised. This brings the lingual aspect of the epiglottis into view, and, always under inspection, the end of the instrument is then pushed gently over the tip of the epiglottis, while the handle is carried still further up, and with it the patient's head undergoes some extension. At the same time, the tube-spatula, making firm pressure on the epiglottis and dorsum of the tongue, brings into view, first the arytenoid region and posterior segment of the larynx, and then the whole length of the cords from back to front, including the anterior commissure. (Fig. 21.) We are now looking through a short, straight and relatively wide tube directly into the larynx, and through the larynx between the cords we can see into the trachea as far down as the carina. When the trachea and bronchi have to be explored, as in bronchoscopy, a longer, finer, tube is used, but the first act of its insertion is the same as that we have just described for direct laryngoscopy. (See p. 166.) At present we are only concerned with laryngoscopy.

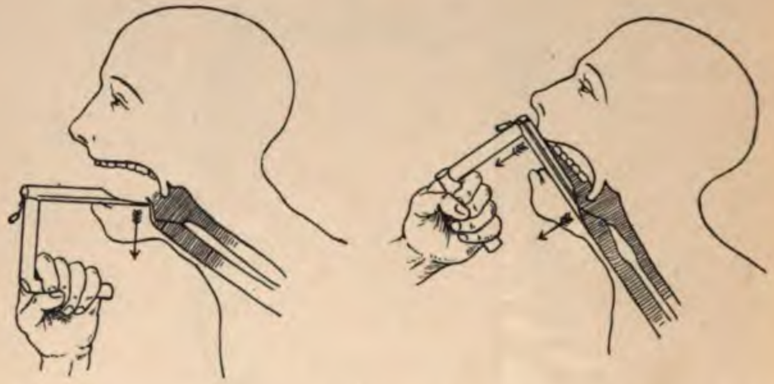

FIG. 21.—Direct Examination of the Larynx (from Chevalier Jackson's "Peroral Endoscopy").

The *direct view* of the interior of the larynx differs considerably from the indirect view as seen in the laryngeal mirror.

The parts seem much nearer and more accessible, and one obtains a clearer notion of the relative distances. Anatomically, of course, the features are the same, although they do not appear to be of the same shape and colour. The cords, for example, seem to have become more fleshy and thicker. The image appears to be upside down compared with the conventional indirect image.

Difficulties.—Irritable and aged patients with short, thick necks are difficult to examine. And prominent teeth with a small buccal orifice render direct examination arduous and sometimes indeed impossible.

In children under a general anæsthetic, the continual flow of saliva and mucus interferes with the view, and calls for constant swabbing and sponging. But this can often be avoided by an antecedent hypodermic injection of atropin (gr. $\frac{1}{200}$).

The direct method in adults is, for the most part, employed for operative procedures. It does not take the place of indirect laryngoscopy for examination and diagnosis, although, as a matter of fact, a doubtful diagnosis may often be cleared up when the larynx is examined directly. It is otherwise in infants and little children, in whom examination with the laryngeal mirror is impracticable. At such early periods, examination with a tube spatula of suitable size is the only method, apart from suspension laryngoscopy, of inspecting the larynx. It is performed under a general anæsthetic, or indeed, without any anæsthetic at all. Cocaine is dangerous in infancy.

Suspension Laryngoscopy (see Fig. 23) is an ingenious method of exposing the interior of the larynx to view, which we owe to Killian.

A special apparatus is necessary, consisting of a " crane " clamped to the operating table, and depending from the crane is a " suspension hook " with mouth-gag, gutter tongue-spatula, and epiglottis elevator.

The patient lies on his back on the operating table, at the head of which the surgeon is seated. The head of the patient, supported by an assistant or on a special support, is over-extended over the end of the table. The surgeon, taking the prepared suspension-hook in his left hand, inserts the spatula into the mouth with the gag closed. Having adjusted the tongue-spatula so as to lie on the tongue, which in this position is uppermost, the gag is fitted to the incisors. The end of the tongue-spatula should reach as far as the glosso-epiglottic fossa. The suspension hook is now hung on the crane, and as this suspends the head, the assistant or his surgeon may release his support of it. The gag is now screwed open, and a ray of reflected light from the forehead mirror thrown into the pharynx shows the epiglottis.

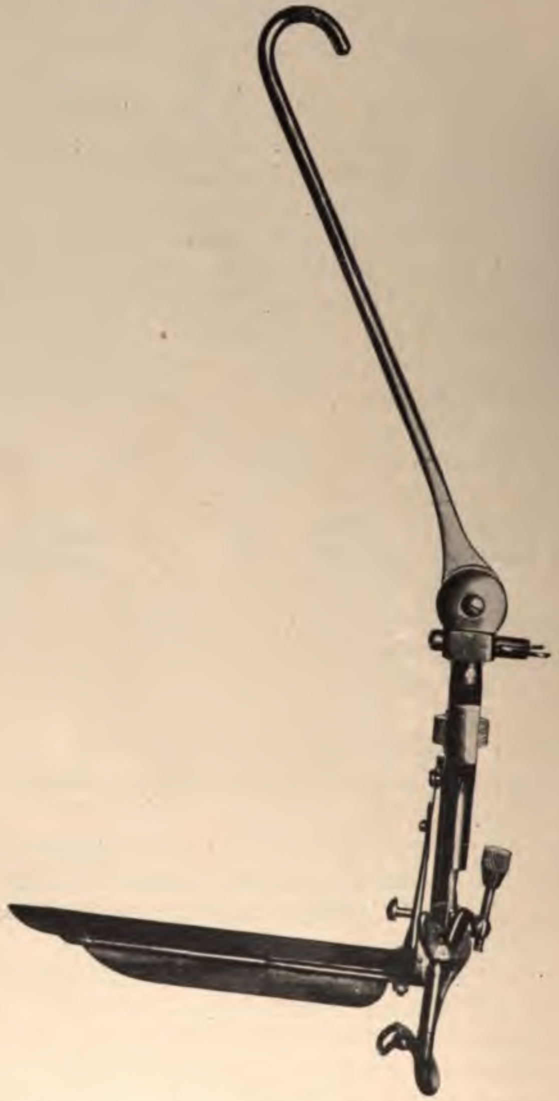

FIG. 22.—Suspension Laryngoscopy—Hook and Tongue Spatula.

The long epiglottis spatula is next inserted and carried over the epiglottis, so as to raise that structure out of the way, and the spatula is then fixed in position by its screw. A few turns of the adjusting screw on the hook will now bring the vocal cords into view, and it is then possible, by means of long probes and forceps, to investigate the interior of the larynx under direct vision. (See Fig. 23.)

Suspension laryngoscopy is chiefly used for the examination and treatment of the larynx in infancy and childhood, and by its means we are able to undertake bimanual manipulation of the larynx. In infancy its use necessitates a general anæsthetic.

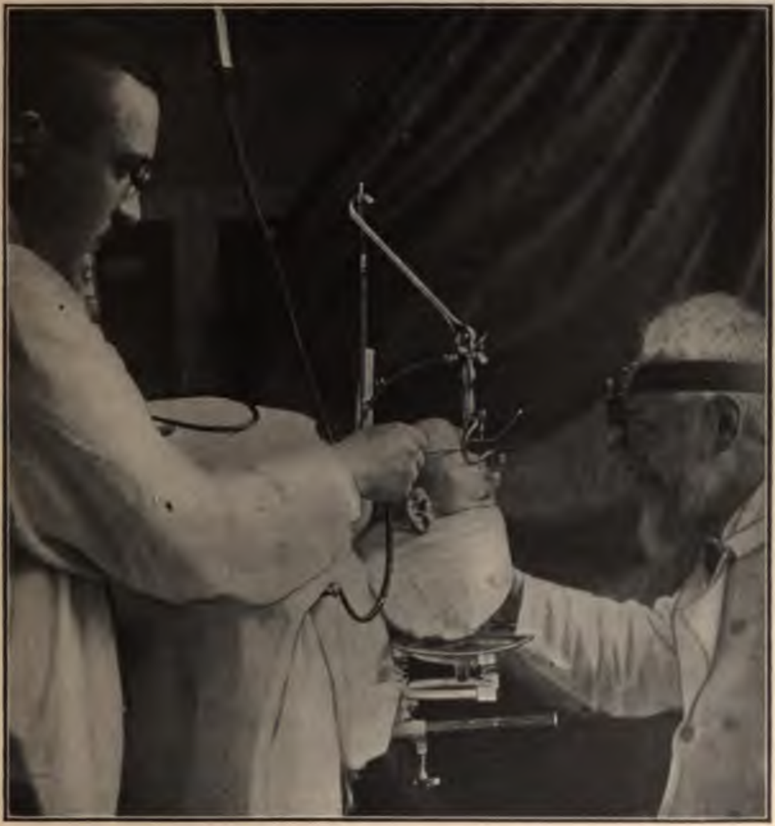

FIG. 23.—Suspension Laryngoscopy (Killian's).

External Examination of the Larynx.—No clinical examination of the larynx is complete without an inspection and palpation of the laryngeal cartilages from without. Many cases also will require an examination of the lungs, heart, and central nervous system.

THE TECHNIQUE OF LARYNGEAL THERAPY

Local Anæsthesia

For the ordinary indirect examination of the larynx, no anæsthesia is, as a rule, required, although a slight spraying of the fauces with 10 per cent. sol. cocaine is sometimes necessary. But the pharynx may be so irritable that even free swabbing with the anæsthetic is insufficient to enable the laryngeal mirror to be held in position.

In such cases, and generally when operative measures are to be undertaken, as in the removal of growths from the larynx, the patient should receive 10 grs. potass. brom. three times a day for two or three days—if time can be obtained, and immediately before the *séance* is begun a suitable dose (gr. $\frac{1}{4}$ to $\frac{1}{3}$) of morphia and atropin (gr. $\frac{1}{150}$ to $\frac{1}{100}$) hypodermically may be given.

The **Routine Local Anæsthetization** of the larynx is gone about as follows:—

A mixture of equal quantities of 10 per cent. sol. cocaine hydrochlor. and sol. adrenalin (1 to 1000) is prepared. Under inspection and illumination a throat swab charged with the solution is passed into the pharynx and applied to the glosso-epiglottic fossæ. In a few minutes the application is repeated, and at the same time the faucial pillars, soft palate and uvula are painted. After another short interval, these parts are again swabbed, together with the posterior pharyngeal wall. The patient should avoid swallowing the solution. (See also p. 21.)

Next, a laryngeal syringe is filled with the solution, and under inspection with the laryngeal mirror, the point of the syringe is introduced over the base of the tongue and over the tip of the epiglottis. While in this position a few drops of the cocaine solution are made to trickle down the epiglottis as down a gutter into the interior of the larynx where the coughing it sets up distributes the anæsthetic freely over the mucosa.

Two or three of such applications suffice to render the larynx anæsthetic. But before starting any manipulation, the laryngeal probe should be passed into the larynx, and its sensibility tested.

The anæsthesia lasts about twenty minutes, and is sufficient for all minor operative measures.

Passing Laryngeal Instruments by the Indirect Method.—The rectangular laryngeal curve renders it advisable to pass these instruments on the flat through the mouth as far as the pharynx, and then to turn the handle and point into the middle line. This is done, of course, under the guidance of the laryngeal mirror.

In operations such as the indirect removal of small neoplasms, many difficulties arise from the nervousness and irritability of the pharyngeal reflexes, but these can be overcome with patience, bromides, or a hypodermic of morphia previous to the examination, together with careful local anæsthetization of the parts.

When a pendant epiglottis obstructs the laryngeal orifice like the half-closed lid of a box, a suture may be passed through it by means of C. Horsford's epiglottis needle-holder (see Fig. 24), the ends of the suture being brought out of the mouth and held by pressure-forceps. The weight of the hanging forceps is sufficient to hold the epiglottis forward.

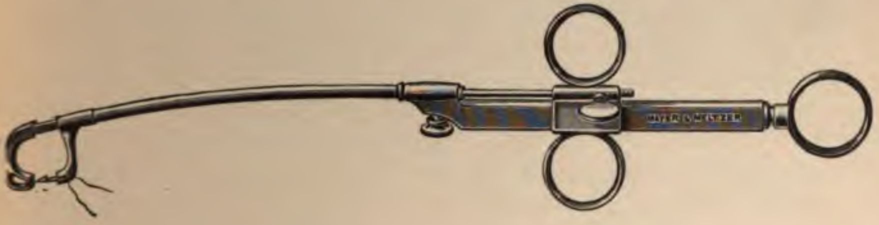

FIG. 24.—Horsford's Epiglottis Needle.

Considerable manipulative dexterity and practice are necessary to become expert at passing laryngeal instruments, and the student should lose no opportunity of acquiring this accomplishment.

By the **Direct Method**, straight, long instruments are used, with a rectangular bend at the proximal end of the instrument. (Figs. 30 and 31.) This method is more rapid and precise than the older method, but the laryngologist must learn to practise both, as there are some patients in whom the use of the direct laryngeal and other tubes is a matter of great difficulty.

The passage of the laryngeal swabs, the application of astringents and of the galvano-cautery point to the interior of the larynx do not introduce any new principle, and require no particular description.

Hypopharyngoscopy.—The following manœuvre, which opens up to the view by the laryngeal mirror of the upper part of the laryngo-pharynx, is known by this name. It is practised when there is any reason to suspect disease of the lower pharynx.

After the larynx has been anæsthetized, a rigid metal probe, guarded by wool wound round the point, is passed under indirect inspection into the larynx as far as the anterior commissure of the cords. By applying steady and forcible traction on the probe, the whole larynx can now be pulled forward away from the posterior wall of the deep laryngo-pharynx, a considerable portion of which is thus made visible in the mirror. Carefully and steadily practised, the method is harmless.

CHAPTER V

AFFECTIONS OF THE LARYNX

FRACTURES AND INJURIES

Fracture of the Hyoid Bone is usually the result of direct violence, as in strangling or hanging. The body or one of the cornua may be broken.

Symptoms.—There is pain on moving the neck, jaw and tongue, and in speaking. The deformity can be felt on palpation, and may be visible in the laryngeal mirror. If the mucous membrane is torn, bleeding into the larynx may occur. Œdema of the glottis is liable to supervene.

Treatment.—The patient is kept in bed, and fed on liquids. Displacement may be rectified manually. If glottic œdema leads to dyspnœa, tracheotomy may be necessary.

Contusions of the Larynx from blows received in boxing or from falls across ropes or other obstacles are not uncommon. There is considerable pain, and the subperichondrial effusion may be evident in the interior of the larynx as well as externally.

But deformity and swelling are more usually noticed when **the thyroid cartilage is fractured,** a serious condition, as it is apt to produce obstruction to the airway either by effusion and swelling in the interior of the larynx, or by the fragment of the broken cartilage becoming displaced.

Symptoms.—The history of the accident is usually clear. Locally, there is pain, tenderness on pressure, swelling, ecchymosis, dysphagia, and sometimes, as has been said, dyspnœa. Emphysema of the neck is a rare but grave phenomenon.

Examination with the laryngeal mirror shows intralaryngeal swelling and sometimes considerable deformity of the interior of the larynx as when there is fracture with displacement, and the vocal cord on the affected side is immobilized. Œdema may develop subsequently.

Treatment.—If there is any dyspnœa ; if shortly after the accident swelling seems to be rapidly forming ; or if there is emphysema, tracheotomy should be at once performed, as urgent symptoms may appear with suddenness, and the patient die from asphyxia before he can be relieved. Otherwise, the pain of simple contusion or uncomplicated fracture may be eased by hot fomentations.

The patient should be kept at rest, and should refrain from using the voice until the pain and swelling subside.

Wounds of the larynx are generally the result of war-injuries, such as bullet-wounds, or of attempts at suicide by cutting the throat.

Bullet-wounds of the larynx, if severe or extensive, so that large vessels are divided, are usually rapidly fatal from hæmorrhage or asphyxia. But if tracheotomy can be promptly performed and the bleeding arrested, the case may be saved.

It is generally with the after-effects that the laryngologist has to deal in the shape of cicatricial stenosis of the larynx. (See p. 163.)

In cut-throat injuries above the hyoid bone, the transverse incision may divide the root of the tongue and sever the lingual, facial, and perhaps the carotid vessels. Apart from hæmorrhage, the danger to life consists in the liability of the epiglottis and tongue to fall back over the larynx, and so to cause asphyxia.

Wounds of the larynx which traverse the laryngeal cartilages may cause death by hæmorrhage or by asphyxia from bleeding into the trachea. If the windpipe is incised or divided, asphyxia may result from its displacement.

The symptoms which appear during the healing of such wounds naturally depend upon their extent and character. Sepsis is common, and the discharges entering the lungs are prone to induce septic bronchitis and pneumonia.

Treatment.—After arresting hæmorrhage, the dyspnœa is relieved by tracheotomy if required, and the wound is sutured in layers, provision being made for drainage.

CONGENITAL LARYNGEAL STRIDOR

This is a form of inspiratory stridor found in new-born infants. Direct examination shows that the stridor is produced by the sucking in of the ary-epiglottic and lateral supra-glottic structures when, as is occasionally the case, they are, along with the epiglottis, congenitally flattened from side to side in such a way as to reduce the orifice of the larynx to a long, narrow antero-posterior slit (Lambert Lack).

Symptoms.—The stridulous noise appears at or shortly after birth, and naturally is a source of alarm to the parents. The sound is of a rough, purring quality, and attends inspiration chiefly, although it may be heard also a little with expiration. It is loudest when the infant is crying or excited. During quiet moments, or while the baby is asleep, it may become entirely inaudible. A certain duskiness, not amounting to cyanosis, may be observed when the noise is at its loudest, and the obstruction induces some intercostal and incisural retraction during inspiration.

The diagnosis from diphtheria and from croup lies in the history and in the chronicity of the phenomenon. Stridor from an enlarged thymus is principally expiratory (St. Clair Thomson).

From papilloma of the larynx, the distinction lies in the fact that the congenital stridor dates from birth, whereas stridor from papilloma is acquired later, but a direct examination of the larynx may be necessary to arrive at certainty.

Prognosis and Treatment.—The symptom continues for two or three years and then gradually disappears, as the child grows, and the larynx becomes larger and roomier.

As a rule, no treatment is required. If any attack is so severe that the child becomes cyanotic, oxygen, if available, may be administered; but asphyxia—even partial asphyxia—does not occur, and the alarming noise can generally be stopped by quietening the child.

ACUTE LARYNGITIS

is most commonly catarrhal in nature, occurring in the course of a " cold," in which case it is due to infection by one of the organisms of acute catarrh.

The inhalation of irritating vapour or dust-laden air and the improper use of the voice predispose to, or may even directly induce the disease. The frequency with which mouth-breathers are the subjects of laryngitis, acute or chronic, should be remembered, and in purulent forms of nasal trouble also, laryngitis may arise. Alcoholism, gout, and rheumatism predispose to it, and it may also appear in the course of measles, influenza, typhoid, or scarlet fever.

The *General Symptoms* in the catarrhal variety are those of a " cold," and in other varieties they depend upon the primary disease.

The *local symptoms and signs* are approximately the same for all, their severity naturally depending upon the degree of inflammation. In the milder cases, hoarseness, dry tickling, painful cough, and a sense of local discomfort, mark the occurrence of acute laryngeal catarrh, and these gradually lessen and pass off in a few days. In the more severe cases there is complete aphonia, the cough is rough and barking, and if œdema co-exist there will be some stridor. The mirror shows general reddening and swelling of the mucous membrane, which is dotted here and there with mucus. The vocal cords lose their flat even contour and become rounded, while their pearly-white colour is replaced by the redness of a hyperæmia which varies in intensity with the degree of inflammation. Even in the milder cases imperfect approximation of the cords on phonation will be evident.

In children, acute laryngitis is both more frequent and more dangerous than in adults. The danger arises from the relatively narrow chink of the infantile glottis, and from the readiness with which œdema and spasm occur. Respiratory distress and croupy cough are more severe than in adults, and in the evening, or during the night, attacks of grave dyspnœa from glottic spasm may suddenly come on, lasting from a few minutes to several hours. These attacks are generally evanescent, but death from asphyxia may occur. In a child, this attack of glottic spasm and acute respiratory distress may be the first symptom of laryngeal inflammation. After the acute dyspnœa passes away, however, the croupy cough and other symptoms of laryngitis remain. This serves to distinguish these attacks of inflammatory croup from the purely spasmodic laryngismus stridulus. A favourable case of acute laryngitis in children runs very much the same course as in adults. After the lapse of a few days, the cough, hoarseness, and fever disappear, and the child gets rapidly well. Some children, however, frequently evince a predisposition to croup every time they catch a cold, a history which should always lead to an examination for adenoids.

Diagnosis.—The sudden onset and associated symptoms render the diagnosis clear, but in children the possibility of primary laryngeal diphtheria should lead to a bacteriological examination of the secretions if the " croupy " signs continue for any longer than twenty-four hours.

Recurrent attacks of acute laryngitis in adults should lead to an examination of the lungs for tuberculosis, and of the nose for nasal suppuration.

Prognosis.—Acute catarrhal laryngitis being not infrequently the first sign of an acute "cold," the prognosis is that of the latter, that is to say, it is good in healthy people but less favourable in aged or debilitated patients. (See p. 258.)

Treatment.—A quick cure may be obtained by a hot bath, retirement to bed between blankets, and a stay there for twenty-four hours. But this ideal cannot always be secured. In any case, however, the patient should remain indoors for a day or two and should give to the voice absolute rest.

The local uneasiness is relieved by steam inhalations containing tc. benzoin co. ♏ 10 to 15 to the pint; or ac. carbol. liq. ♏v to the pint of nearly boiling water.

Hot drinks of lemonade, barley water, and gruel are very grateful.

A day or two after the acute symptoms have subsided, the patient is able to go out of doors unless the weather is inclement, but care should be exercised, especially by singers, not to use the voice too early.

If signs of extension into the chest are present, however, further confinement to an equable indoor temperature will be necessary.

In children, croup requires special treatment. The child is put into a hot mustard bath and wrapped in flannels. For the paroxysms, a sponge wrung out of hot water and laid on the front of the neck often affords relief. Care must be taken, especially in young infants, to avoid scalding the skin. Internally the emetic action of a teaspoonful of ipecacuanha wine is useful, and after that has passed off, small doses of p. ipecac. co. prove a useful sedative to the irritated larynx. The drug must be administered cautiously, however, as the therapeutic action of opium in children, though valuable, may entail risk. It is unsuited to babies of less than a year old.

If the breathing becomes seriously impeded in children, intubation or tracheotomy may be necessary. (See p. 178.)

ACUTE INFLAMMATORY ŒDEMA OF THE LARYNX

The submucous tissue of the larynx, both above the vocal cords and below them, is looser than it is in the cords themselves, and for that reason these parts are liable when inflamed to become so swollen and œdematous as to lead to serious interference with respiration. Although in this process the cords themselves are, as a rule, but slightly affected, it is a mistake to say that they do not become œdematous at all.

The condition is commoner in children than in adults, as it is, indeed, the pathological change underlying genuine inflammatory croup. But it is not unknown in adults, and it is, at any age, a source of danger to the patient.

Etiology.—Acute inflammatory œdema of the larynx may result from injury—mechanical, thermal or chemical. It is produced also by an invasion of the tissues by the pneumococcus, and other pyogenic organisms, and it is not infrequently secondary to syphilitic, cancerous, or tuberculous laryngeal disease. It is also an associate of septic pharyngitis, Ludwig's angina, and similar conditions.

The symptoms are those of acute catarrhal laryngitis—cough, hoarseness, and pain on coughing; but there is more constitutional disturbance, while dyspnœa with stridor, more or less severe, is almost always present, from the obstruction to the breathway presented by the swollen and œdematous epiglottis, arytenoid region, and glottis. The obstruction may be so great as to cause death by asphyxia.

As we have just seen, acute inflammatory œdema of the larynx may arise in children from simple acute laryngitis, and it forms one of the varieties of "croup." Among other diseases which give rise both in children and in adults to what is called "croup," or laryngeal stridor, are simple (or septic) membranous laryngitis, diphtheria of the larynx, spasm of the glottis, and a foreign body arrested in the larynx. (See also p. 9.)

In adults, the laryngoscope, in acute inflammatory œdema of the larynx, shows the epiglottis puffed up and swollen out of all resemblance to its normal condition. Instead of being pale, yellow, flat, movable, and leaf-like, it is immobile, tense, and sausage-shaped, and varies in colour from purplish-red to pale pink. The characteristic translucency of its margins proclaims the distension to be due to œdema. If, as is generally the case, the ary-epiglottic folds, the arytenoid eminences and the inter-arytenoid region also take part in the œdema, the vestibule of the larynx may be so encroached upon by the puffy swelling as to prevent any view of the glottic or subglottic region being obtained.

Occasionally, the course of events is such that the œdema shows a travelling tendency, and we may in the course of a few days or weeks find it leaving the supra-glottic for the glottic, and then for the infra-glottic region. In adults, at the same time it is usually possible to make out a certain amount of loose œdematous infiltration of the tissues in the neck external to the larynx. The process does not extend to the trachea.

In children, the excitement and general distress of " croup " render it impossible for the surgeon to use the laryngoscope, so the condition of the larynx can only be inferred from the symptoms.

The œdema causes dyspnœa and noisy stridor. If there is any cough, it is rough and " croupy," and the voice is reduced to a whisper.

The dyspnœa varies in degree with the amount of obstruction to the airway in the larynx, but its presence always induces a feeling of anxiety in the patient, and the dyspnœa is, moreover, liable to sudden exacerbations from rapid increase in the exudation or from uncontrollable spasm.

The pulse varies in strength and rapidity according to the nature of the infective agent. When rapid, compressible, and " shabby," the outlook is bad as the case approximates to the Ludwig type. (See p. 41.) On the other hand, if the pulse is not unduly increased, and is of good volume, the chances are favourable—so long as respiration can be kept going.

Diagnosis—A culture, from the larynx if possible, should in all cases be taken in order to exclude diphtheria.

That the œdema in adults may be induced by some hidden tuberculous or malignant lesion should never be forgotten, and a search should be made for those diseases after the œdema has subsided. Acute œdema of the larynx may also prove to be the first sign of perichondritis. (See p. 109.)

Prognosis.—As long as the œdema exists there is anxiety, and death may be sudden. The stridor and dyspnœa may manifest considerable fluctuation in severity, but a progressive improvement is a hopeful augury of entire recovery in simple uncomplicated cases.

The pneumonic type may extend to the lungs, setting up broncho-pneumonia, and may thus initiate a prolonged and anxious illness.

Treatment.—When mild, the application of cold packs to the neck with the sucking of ice will generally suffice to relieve the symptoms. Sometimes, however, the patient prefers warm applications and drinks.

A throat or laryngeal spray of cocaine (1 to 2 per cent. in water) with an equal quantity of adrenalin solution (1-1,000) every two or three hours will help to reduce the swelling.

Even when the disease is mild, preparation should always be made for immediate *tracheotomy*, as the necessity for operation may arise suddenly. In any case, if the stridor is considerable, and especially if it is increasing, the operation should be performed before urgency calls for immediate and hasty interference. It is best performed under local anæsthesia. (See p. 180.)

Non-Inflammatory Oedema of the larynx is met with in Bright's disease; as a result of passive hyperæmia from pressure on the veins of the neck; and sometimes in consequence of the administration of potassium iodide.

The œdema has the same distribution as that met with in acute inflammatory conditions, but the mucous surface is pale instead of red or purplish, as it is in the acute œdema.

The risk of laryngeal œdema when Potass. Iodid. is administered for syphilis of the larynx is considerable, and prior to the days of the salvarsan drugs, it was often necessary, on that account, to perform tracheotomy before beginning the iodide. One or two preliminary doses of the arsenical agent, however obviate that necessity. (See p. 115.)

CHRONIC HYPERPLASIA OF THE PHARYNX AND LARYNX

Under this non-committal name is described a rare and peculiar condition of semi-solid infiltration of the submucous tissues of the pharynx and larynx, comparable in some respects with amyloid disease, but of unknown nature (Logan Turner).

It produces a general, smooth fullness of the parts and interferes with their movement, but apart from this inconvenience it exercises little or no deleterious influence.

It is extremely chronic, lasting unaltered for years, but it may undergo improvement and even entirely disappear as mysteriously as it came.

No treatment has any effect upon the condition.

MEMBRANOUS LARYNGITIS

Membranous Laryngitis may be septic or diphtheritic, but the latter is by far the most common. When first seen, these varieties can only be differentiated bacteriologically, and even later on in the course of the disease, at all events in young children in whom laryngoscopic examination is difficult or impossible, diagnosis from clinical evidence alone should never be made.

In both forms of the disease the signs of local interference are the same—they are those of laryngeal obstruction, together with hoarseness and croupy cough. There is present also constitutional disturbance—headache, fever and prostration. After the first day or two, the signs of obstruction become more marked, attacks of glottic spasm come on, and the stridor increases. The child is now said to be "crowing," and suffering from croup. We should always remember that **croup is not a disease : it is only a symptom.** In membranous laryngitis, however, croup is a more marked symptom than in any other form of laryngeal disease.

The constant and distressing cough—which, when paroxysmal, is apt to pass into spasm—the dyspnœa and lividity, the constant tossing about of the child in its frantic efforts to obtain more air, the clutching at the throat, and, as time goes on and no relief to the obstruction is afforded, the gradual onset of lethargy, and at last of coma from carbonic acid poisoning, unite to form one of the most distressing sights a doctor can be called upon to remedy.

In adults the attack is less menacing, as a rule. The presence of the membrane only produces symptoms of laryngitis, and the diagnosis from other forms of laryngeal obstruction is more readily made, since a satisfactory examination with the mirror is easily obtained.

Dyspnœa in adults is less frequently observed, but when it does appear, the prognosis is apt to be worse than in children, as its presence in adults indicates a relatively more serious attack.

If membranous *pharyngitis* is associated with the croup, we can argue with fair certainty that the disease in the larynx is diphtheria; and when membranous casts of the larynx or trachea and bronchi are coughed up, the same diagnosis may be made clinically. But in all cases of membranous laryngitis, as in membranous pharyngitis, the aid of the bacteriologist should be sought to enable us to base our diagnosis upon a sure foundation. (See also " Diphtheria," p. 37.)

Prognosis.—If the disease proves to be diphtheria, the prognosis will be that of that disease.

We may say, however, that laryngeal diphtheria, whether primary or secondary, whether in children or in adults, always renders prognosis grave.

Treatment.—Locally, the treatment is the same as for acute laryngitis with œdema. Tracheotomy or intubation may be called for early in the case.

Generally, the treatment will be dictated by the nature of the infection.

CHRONIC LARYNGITIS

Under this heading, a large number of diseases must be gathered, some of which have nothing in common with each other save that they induce chronic inflammation in the larynx. Resembling each other closely in their laryngeal manifestations, they supply us with the diagnostic puzzles of laryngeal practice.

Even if we exclude tuberculosis, lupus, syphilis, and cancer, any of which may induce appearances simulating simple chronic laryngitis, the varieties of the disease are very numerous, ranging as they do from the chronic hyperæmia of overworked singers to the pachydermia of alcoholism and Bright's disease.

Etiology.—Chronic laryngitis can be induced by abuse of the voice, a variety of the disease at once common and difficult to cure. Otherwise, it is practically always secondary to some other abnormality or disease.

It may follow a series of acute or subacute attacks of catarrhal laryngitis. It is frequently set up by irritating dust, such as the chalk dust of the school black-board; or by irritating gases or vapours, as in warfare. It is common in alcoholics, and is liable to come on as a result of excessive smoking, especially if the habit of inhaling is practised.

The pachydermatous type is often part and parcel of a constitutional tendency to fibrosis, which may also be expressed in albuminuria and other phenomena of chronic Bright's disease (Jobson Horne).

But of all the factors which tend to the production of chronic laryngitis, it is generally agreed nowadays that the most frequent is some abnormality or disease in the nose, such as: a deflected septum with chronic nasal catarrh; nasal sinus suppuration; and atrophic rhinitis.

Finally, although we can only deal with them in their proper place, it must not be forgotten here that tuberculosis, lupus, syphilis, and cancer all induce by their presence more or less chronic laryngitis, and consequently in their earlier stages they may give rise to appearances in which no indication of any grave underlying disease is manifest.

Pathology.—The changes start with hyperæmia and lymphocytic infiltration of the mucous and submucous layers of the larynx and pass on to a general fibrotic hypertrophy of the soft tissues. The mucous membrane is hyperæmic, thickened, and thrown into folds; the vocal cords become red and thick; and the formation of outgrowths in the shape of fibromata and other tumour-like projections in or about the cords is common.

Symptoms of Chronic Laryngitis.—More or less huskiness of voice exists, especially after prolonged use, as in public speaking or singing. Or the voice gets quickly tired, and is lost altogether after a little use. There is frequently dry cough, with, at times, a continual tickling in the pharynx or larynx. Another common symptom of the early stages, and one very annoying to public singers and speakers, is the accumulation of mucus in the larynx, and the frequent necessity felt to "clear the voice," sometimes in the middle of a sustained note, or at other inconvenient moments. (This symptom is often complained of before any change in the tone of the voice is observed. Not infrequently it is due to chronic nasal catarrh. Sometimes no cause can be found for it, and no sign of any disease either in the larynx or in the nose can be observed.)

In general, unless the patient is a singer or public speaker, the earlier symptoms of chronic laryngitis are overlooked or ignored, and by the time the patient comes to consult us the second stage of the disease has been reached, in which definite hoarseness is present. The voice is now rough and breathy, or husky, and shows a tendency to complete aphonia after it has been resting, as in the morning. The easy flexibility and power of varying the pitch and strength of the voice is diminished even for ordinary conversation, and singing can no longer be practised. Coughing is usual, especially after getting up in the morning, and the noisy, hawking efforts of the patient struggling to get rid of an imaginary accumulation of " phlegm," aggravate the already existing laryngeal irritation.

The third stage consists of more or less permanent aphonia. Many patients never get as far as this third stage ; others, again, arrive rapidly at the state of aphonia by reason of the development of a tumour on one or other cord interfering with their closure.

On *examination*, the appearances vary with the stage and severity of the disease.

At first, little or nothing may be found to correspond with the patient's complaints. Or the pink colour of the cords may be deeper and they may appear a little fuller than usual.

These appearances are more pronounced immediately after the voice has been used, but they tend to become permanent, until finally the cords become uniformly red and infiltrated, with thickened, rounded edges, presenting on attempted phonation more or less imperfect approximation, there being a fine elliptical opening between them as they vibrate.

In the third stage, the cords are thick, deep red, and fleshy, and no longer seem to differ either in colour or in texture from the rest of the interior of the congested larynx. Their movements are sluggish, their approximation imperfect, and their surface is dry. At this period a certain amount of thickening and protuberance of the interarytenoid mucosa is common, and this being thrown into prominent folds during attempted phonation still further impedes the proper closure of the cords, particularly towards their posterior ends. This condition is sometimes known as **hypertrophic laryngitis**.

Along with these changes, there is often increase in the quantity of mucus secreted, and it tends to become thick and sticky, and may be seen adhering to the cords.

Occasional shallow breaches of the surface of the mucous membrane will be noticed, especially towards the posterior end of the cords.

The following sub-varieties of chronic laryngitis are distinguished :—

Pachydermia Laryngis.—This name, introduced by Virchow, has become a mere term of convenience ; it is now generally limited to those cases, by no means uncommon, where the vocal cords, especially towards their posterior ends, manifest thickening, redness and irregular but symmetrical, blunt elevations or prominences. The condition may be discovered by accident, as symptoms may be entirely absent, but as a rule more or less interference with phonation is present, and sometimes, indeed, the voice is very rough and hoarse.

On examination, red and flesh-like prominences on the vocal processes are visible, and the typical clinical picture presents itself when the prominence in the one cord corresponds to a depression in the other, into which it fits during phonation.

The appearance is very liable to be mistaken, sometimes even by experienced eyes, for malignant disease. The great distinguishing feature is that pachydermia is symmetrical, whereas epithelioma is unilateral.

In like manner it may be mistaken for syphilis or tuberculosis. On the other hand, either of those two diseases may produce pachydermia.

Nodular Laryngitis.—Chorditis tuberosa—(Singers' Nodes).—In this form of chronic laryngitis, which is due to faulty voice-production, to overstrain or other abuse of the voice, two small nodules appear on the edge of the cords at the junction of the anterior and middle third, and opposite to each other. By interfering with the effective closure of the cords, they induce persistent hoarseness. (Fig. 25.) An analogous change is responsible for the aphonia sometimes observed in crying children (Screamers' Nodes).

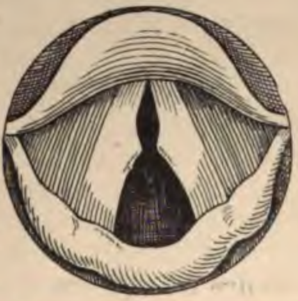

FIG. 25.—The Situation of Singers' Nodes (diagrammatic).

If the nodules are sufficiently prominent, they may be removed by suitable forceps, either by the direct or by the indirect method. Otherwise they will disappear if absolute silence can be maintained for a prolonged period. After their disappearance, measures must be adopted to prevent their recurrence in the avoidance of the faults in voice production that induced them. We must add that singers' nodes may be found in a larynx otherwise healthy and free from inflammatory changes.

Laryngitis Sicca.—(See Ozænatous Rhinitis, p. 282.)

Diagnosis of Chronic Laryngitis.—The presence of the signs of chronic laryngitis should lead to an extended examination of the patient in order to exclude nasal disease; syphilis of the larynx; Bright's disease; alcoholism; and tuberculosis of the lungs or larynx; before we conclude that the case is one of simple or primary chronic laryngitis.

From malignant disease it is distinguished, as we have already observed, by its local symmetry, and also by its persistence with little or no alteration in the signs for prolonged periods of time. It must not be forgotten, however, that cancer may develop in a larynx which is already the seat of chronic inflammatory changes.

Prognosis.—Simple chronic laryngitis will get well spontaneously if the cause can be removed and the voice rested.

The most obstinate cases are those showing pachydermia, especially if the patient be a man of middle age addicted to tobacco, alcohol, and coarse laughter, with a high blood pressure and a low moral code.

The laryngeal changes which would render a soprano or a tenor hoarse or even voiceless, may have little or no influence upon a contralto or a bass, save, perhaps, to deepen the voice. Cases have been recorded (C. Horsford) in which cure of chronic laryngitis has transformed a voice from bass to tenor.

Treatment.—The most probable cause should be sought for and removed. Nasal obstruction or suppuration should be treated; and bad habits reformed.

Locally, the voice should obtain absolute rest for several weeks or months, no public speaking and no singing being permitted. This is often a counsel of perfection, and people who live by their voice will not carry it out.

In obstinate cases, laryngeal aqueous sprays containing some alkali, such as

P. Sod. Bicarb.	gr. v.
P. Sod. Bibor.	gr. v.
P. Sod. Chlor.	gr. v
P. Sacch. Alb.	gr. x.

in 4 oz. of warm water; or oily sprays containing menthol (grs. iii to ℥i); ol. eucalypt.: (♏iii to ℥i) may be employed.

Applications at intervals by means of a laryngeal swab are often useful, such as zinc. chlor. in aq. (10 grs. to ℥i); or

R.	Liq. Ferr. Perchlor.	♏vi.
	Menthol,	gr. iii.
	Glycerin	ad ℥i.

Internally, tonics containing nux vomica or strychnine are useful. But for the cure of the hypertrophic or pachydermatous varieties, potass. iodid. grs. 4 to 6 t. i. d. for prolonged periods has a well-deserved reputation, even when the disease is non-syphilitic in origin.

Singers, clergymen, and other voice-users are frequently the subjects of a *hyperæsthesia laryngis*, and then moral treatment is as valuable as local applications to the larynx, but the latter may be employed as an aid to the former. In any case, the local agent should be mild.

Occasionally, singers with acute catarrhal laryngitis demand a voice for immediate use. For this purpose, a laryngeal spray of adrenalin (1-5,000) is usually administered, but it is apt to be followed by severe reaction, and the risk of permanent injury to the voice is not inconsiderable.

Young persons who are training to become singers, frequently come asking for relief from the early symptoms of chronic laryngitis. The throat-surgeon, consulted by these patients, must use his own judgment. Some require an operation on nose or throat. Some require a change of singing-master or method. Some are not fitted to become vocalists.

Modern methods of teaching and voice-training too frequently endeavour to force from the voice a compass and a sustained strength quite beyond its powers.

Gouty Laryngitis.—The rough or " port-wine " voice of gouty people is due to a general laryngeal congestion with some tendency to œdema of the arytenoid eminences, especially distinct in the morning hours.

The reddish or livid colour of the mucosa serves to distinguish the œdema of gout from that of tuberculosis.

Laryngeal phenomena are frequently associated with gouty pharyngitis. (See p. 48.)

CHONDRITIS AND PERICHONDRITIS OF THE LARYNX

Chondritis and Perichondritis are almost invariably secondary to tubercle, syphilis, malignant disease, typhoid fever, small-pox, diphtheria, or to sepsis, as from the presence of a foreign body in the larynx. Any one or more than one of the cartilages may be affected, but the arytenoid is perhaps most frequently attacked, in which case the corresponding cord is fixed. As a result of the inflammation, a larger or smaller portion of the cartilage dies, and an abscess forms, which may be seen projecting into the larynx, or may be felt in the neck as a tender swelling over one or other cartilage. After the abscess has broken, and the pus is discharged, the sequestrum may be got rid of later. Many cases, however, stop short of necrosis, but in all cases the resulting cicatrization is liable to produce permanent hoarseness or dyspnœa from distortion of the parts, fixation of the cords, etc. It may thus be possible to recognize, on examining the larynx, the traces of a long-past perichondritis.

The *Symptoms* vary with the acuteness of the attack and the character of the primary disease. There is always more or less severe pain, generally with fever, and constitutional disturbance. And if an abscess forms, the swelling and infiltration interfere both with phonation and with respiration. The dyspnœa may be so severe as to necessitate tracheotomy.

In later stages, the laryngeal lumen may be encroached upon by such consequences of the perichondritis as fixation of the cords in the middle line ; the impaction or displacement of sequestrated pieces of cartilage ; cicatricial contraction and adhesions ; and so on.

Prognosis.—The prognosis of laryngeal perichondritis is always serious but varies in gravity with the cause. If the primary disease is cancer or tuberculosis the onset of perichondritis frequently introduces the terminal stage of the disease.

Diagnosis.—The diagnosis of the disease itself presents little difficulty, the pain, often with external tenderness, and the irregular internal swelling with the constitutional effects of a septic process being sufficient to establish the presence of perichondritis. But it is very difficult, and indeed, during the acute stages it may be impossible, to determine what the cause of the perichondritis has been, and this is particularly liable to happen when the causative disease has been latent until the onset of the inflammatory lesion, so that our view of the interior of the larynx is so obscured by œdema and swelling that it is impossible to see whether or not any surface ulceration is present.

Correct diagnosis in the case of tuberculous perichondritis is, however, often rendered possible by the pallid œdema of the interior of the larynx, as well as by the presence of signs of tuberculosis elsewhere.

In doubtful cases, an X-ray examination may give valuable help. The larynx, as well as the thorax, should be skiagraphed, as the perichondritis may be due to a foreign body.

Treatment.—The patient is kept in bed, and hot fomentations are applied to the neck. Internally, ice is given to suck, and opium is useful for the relief of the pain and distress. Œdema may be countered by a laryngeal spray of cocaine (2 per cent.) and adrenalin (1-1000). If an abscess forms and points, it should be opened as early as possible, either externally or internally, according to its situation. The occurrence of dyspnœa should lead to prompt tracheotomy.

These measures having been taken, we proceed to search for the cause, and direct our treatment to its removal or cure.

Inflammation of the Crico-Arytenoid Articulation.—Synovitis of the crico-arytenoid articulation is not uncommon in acute rheumatism, in which disease it may be the first, and, indeed, it is said, the only joint attacked. Gout also is said to affect this articulation at times.

Symptoms.—There is pain deep in the neck, usually on one side only, aggravated by swallowing and sometimes on phonating. The voice may be weak. In the course of a few days the synovitis subsides, and the symptoms are relieved, but there is a possibility of ankylosis of the joint supervening, in which case the voice may be seriously and permanently impaired.

Ankylosis of the Crico-Arytenoid Articulation.—*Etiology.*—Any deposit, gouty or rheumatic, in or around the joint may lead to its fixation, as may cicatrices from wounds such as cut-throat, or from traumata. It may be set up by acute perichondritis from whatever cause arising, and it may be a sequel to the acute inflammatory lesions of septic pharyngitis, diphtheria, scarlet or typhoid fever.

Symptoms.—The fixation of the arytenoid cartilage produced by ankylosis immobilizes the corresponding vocal cord, and thus simulates fixation of the cord from paralysis.

Ankylosis may fix the cord in any position, and upon the position assumed depends the nature of the interference with phonation. The ankylosis may be bilateral, in which case both arytenoid cartilages, and, of course, both vocal cords will be immobilized.

When one cord only is affected, and it is fixed in the middle line, no interference with phonation will be experienced. These cases are, as a rule, accidentally discovered on making a routine examination of the larynx.

In many cases, however, there is considerable dysphonia or actual aphonia, and the patient comes complaining of this disability. Then examination reveals one or other, or, in bilateral cases, both cords fixed in the cadaveric position, more rarely in the position of abduction.

If both cords are affected, and in any position save the median, absolute aphonia will be present. If they are both fixed in the middle line, they obstruct respiration, and may induce fatal asphyxia, although phonation is unaffected. In this position, paralysis of abduction affecting both cords is simulated.

Diagnosis.—Fixation of one or both cords from ankylosis has to be differentiated from fixation of the cord in consequence of paralysis.

In ankylosis there may be some swelling around the affected joint, as may be easily seen by practising hypopharyngoscopy. (See p. 93.) But the simplest method of ascertaining whether or not the arytenoid cartilage is movable is to pass the direct laryngeal tube-spatula, and to endeavour to move the affected arytenoid by means of a stout long probe. If there is ankylosis, the arytenoid will be immovable. If the cause is paralysis without ankylosis the arytenoid will move with the probe (William Hill).

Treatment.—If there is any interference with the respiration from malposition of the cords, a low tracheotomy should at once be performed, and the patient should wear a tracheal cannula for the rest of his life. It may be possible, and it is sometimes advisable, to reconstitute the laryngeal lumen by removing both of the vocal cords by operation, performed with the aid of suspension laryngoscopy. But this measure permanently destroys the voice, and the patient will probably prefer to keep his voice even at the cost of wearing a tracheotomy tube.

SYPHILIS OF THE LARYNX

Syphilis does not attack the larynx so frequently as it does the pharynx, but it is not a rare laryngeal disease by any means, and the laryngologist should be able to diagnose laryngeal syphilis promptly as well as to treat it efficiently.

Hereditary Syphilis may be found affecting the larynx in the form of (1) laryngeal catarrh—the rough, harsh voice of the syphilitic infant—or in (2) tertiary deposits, usually appearing about puberty.

Acquired Syphilis may induce laryngeal disturbances at any period of the disease, these disturbances varying in severity from simple catarrh to deep ulceration with perichondritis, which, if not quickly controlled, may cause death.

(1) **Syphilitic Catarrh of the Larynx** is an early secondary manifestation. It takes the form of persistent erythema, but unless the cords are attacked, it may cause no symptoms and attract no notice.

(2) **Mucous Patches** are rare in the larynx, but they are occasionally seen as areas of greyish thickening on the cords or on the lingual surface of the epiglottis. They may pass into slight superficial ulcers, but the tendency to deep ulceration, so common in the pharyngeal mucous patch, does not seem to occur in the larynx.

(3) **The Gumma** and **Gummatous Infiltration** is the most frequent form of laryngeal syphilis. Before they ulcerate, gummata appear as smooth, rounded, reddish-yellow swellings, which may form anywhere in the larynx, and sometimes produce a more or less general infiltration. As isolated deposits, they prefer the epiglottis, the ary-epiglottic folds, the posterior wall, and the ventricular bands. The ulcer resulting from the breaking down of a gumma is deep and punched out, with abrupt edges and a red areola. And the base is occupied by a fawn-coloured or " wash-leather " slough, which is very characteristic. The ulceration is often accompanied with perichondritis, in which case, pain, external swelling and tenderness, together with constitutional disturbance, bring the case within danger-limits. Necrosis of the affected cartilage with exfoliation occurs in grave and neglected cases, but when promptly treated the perichondrial inflammation tends to resolve quickly. Its healing is productive of considerable cicatricial deformity.

(4) Another typical syphilitic manifestation in the larynx is the **diffuse infiltration,** in the course of which the submucous tissue of the whole interior becomes the seat of small-celled infiltration which induces a general pachydermatous appearance with fleshy protuberances in the inter-arytenoid region, the cords participating in the general hyperplasia.

(5) All tertiary changes in the larynx, when they undergo recovery, eventuate in **cicatricial contractions,** whereby great deformities, irregularities and functional disturbances may be produced. The diffuse infiltration we have just mentioned

for example, when it undergoes healing transforms the whole of the interior of the larynx into cicatricial tissue, and may induce a *fibroid contraction* of the organ. The chink of the glottis may become more or less obstructed by webs of connective tissue stretching from wall to wall; ankylosis of the crico-arytenoid articulation may produce irremediable hoarseness; while the ventricular bands, the vocal cords, and the epiglottis may be altered out of all recognition.

The *symptoms*, of course, depend upon the character and site of the lesion, but hoarseness is the most usual complaint, while cough, and, in the obstructing lesions, dyspnœa may be prominent. Pain is seldom experienced unless perichondritis has been set up.

The *diagnosis* of the nature of the lesion is sometimes difficult.

Gummatous infiltration and ulceration, especially when unilateral, and in the false and true cords, often very closely simulates epithelioma. In syphilis, however, the rate of tissue invasion and destruction is more rapid than in epithelioma. In other words, the history of the case will show us that the symptoms are of recent origin in spite of the relatively wide extent of the disease.

A history of syphilitic infection or a positive Wassermann reaction, while they inform us that the patient has had syphilis, do not, it must be remembered, certify that this or any particular lesion is syphilitic. Cancer may appear in a syphilitic patient. In some cases, therefore, uncertainty may be unavoidable until the therapeutic test of anti-syphilitic remedies has been applied. And even here caution must be exercised, as the exhibition of potass. iodid. in cancer is commonly followed, for a few weeks at least, by a reduction in the swelling and infiltration of the growth.

Some authorities decry the removal of a small portion of the suspected tissue for microscopic examination, but where time is of value, the writer believes it wiser, in a doubtful case, to remove a piece from the diseased area in the larynx for diagnostic purposes than to risk a decision upon naked-eye appearances alone.

From tuberculosis of the larynx, syphilis, as a rule, is easy to distinguish, and attention to the description of the two diseases given in the text ought to be sufficient to prevent errors being made. (See p. 116.) In brief, the infiltration produced by tuberculosis is pallid, and its ulceration, when present, is more superficial, while the granulations are pale and languid. There are usually also indications of tuberculosis in the lungs or elsewhere, while the action of anti-luetic remedies in a tuberculosis case is productive of harm.

Real difficulty indeed seldom arises save in a mixed case; where, that is to say, the larynx is the seat of combined syphilis and tuberculosis, and this possibility should always lead to an examination of the lungs in a doubtful case.

The *prognosis* of syphilis of the larynx under treatment is good as regards life, except in advanced cases, where perichondritis has had time to develop. But the recovery of a clear voice can scarcely be expected if the cords have been attacked by the disease. At the same time, the amount of recovery of voice obtained by suitable treatment is often surprisingly great.

Where the larynx has undergone extensive and obstructive cicatrization, a tracheotomy tube may have to be constantly worn.

Treatment.—Before the introduction of the salvarsan group of remedies, it was often found advisable to perform tracheotomy before beginning treatment with potass. iodid., as this drug frequently induces glottic œdema, and so may imperil the patient's life. But nowadays, even where there is already a certain amount of stridor present, tracheotomy may be dispensed with if salvarsan be given, as the improvement that drug effects upon the larynx is so immediate and considerable that stridor is rapidly lost.

Tracheotomy, however, may nevertheless still be necessary when the obstruction is of such a character as to resist the anti-syphilitic remedies, as when a perichondritic swelling or a cicatricial contraction or webbing has blocked up the lumen of the larynx.

Such adhesions and webs, however, may be divided intralaryngeally with benefit, although the result, it should be added, is not always up to expectation. They are very prone to re-form.

Locally, attention is applied to the cleansing of septic ulcers with sprays, as of hydrogen peroxide (10 vols. per cent.). If there is pain on swallowing, it may be relieved by insufflations or inhalations through Leduc's tube of orthoform or anæsthesin. Exuberant granulations may be curetted, but under vigorous general treatment they disappear spontaneously.

For *general treatment of syphilis*, see p. 69.

It is a wise plan if there is stridulous breathing to confine the patient to bed until the dangerous period has been tided over, and arrangements should be made for the immediate performance of tracheotomy if the necessity for urgent interference should arise. But it is less likely to do so than in the more severe types of inflammatory, diphtheritic, tuberculous, or cancerous obstruction, at all events once salvarsan has been administered.

TUBERCULOSIS OF THE LARYNX

(Tuberculous Laryngitis ; Laryngeal Phthisis)

Tuberculosis is one of the most important laryngeal diseases by reason of the facts that (a) its presence may render certain the diagnosis in a case of suspected pulmonary tuberculosis ; (b) its influence upon the prognosis of phthisis pulmonalis is very powerful; and (c) the treatment must be directed to the laryngeal as well as to the pulmonary lesion, although it is no doubt true that the disease in the larynx frequently gets better or worse in harmony with the condition of the lungs, and of the general status of the patient.

Pathology.—The disease is practically always secondary to pulmonary tuberculosis, and if it appears in any case in which pulmonary deposits cannot be found clinically their presence may nevertheless be assumed from the fact of the laryngeal disease.

For that reason it has been considered probable that the laryngeal disease is induced by sputum infection, and the local incidence of the tuberculous deposits supports this view, seeing that they affect, in the first instance at all events, the posterior regions and those parts, such as the arytenoid and inter-arytenoid regions, where sputum is liable to cling and collect.

Some pathologists, however, favour the opinion that the infection is borne to the larynx in the blood and lymph vessels from the lungs, because it is a submucous lesion to start with, and because it often develops in cases of pulmonary tuberculosis in which there is no expectoration.

The pathological process does not differ from that of tuberculosis elsewhere. There is the stage of tubercle formation in and around bacillar groups, followed by caseation-necrosis with breaking down, and in the superficial parts the formation of ulcers. In the larynx, the arytenoid and inter-arytenoid regions are the most liable to the disease. The next in order of frequency are the vocal cords; the ventricular bands; and lastly, the epiglottis. But ulceration is most common on the cords and least common on the arytenoids (Sir St. Clair Thomson).

Healing, when it takes place, is brought about by lymphocytic infiltration and by fibrosis. Some cases manifest a strong tendency towards fibrosis and cure from the outset, while in others little or no success seems to attend the tissue-reaction against the bacillus.

For the sake of convenience, the progress of the disease may be described in stages or periods, but in most cases these stages blend and coincide. These are the stage (1) of infiltration, (2) of ulceration, (3) of perichondritis. Fibrosis may attend any one of the stages, but the most favourable cases do not progress beyond infiltration with a little superficial ulceration. The less favourable show deep ulceration with profuse granulations, and the least favourable a general grey infiltration of the whole interior of the larynx, rapidly breaking down without any evidence of fibrous tissue formation.

Symptoms.—Tuberculosis of the larynx may set in without producing any symptoms referable to the larynx. Cough is often absent, and when it is present, it is pulmonary rather than laryngeal in origin. The voice may manifest no change at all at this early date. The fact of this latency, therefore, renders an examination of the larynx an *important routine procedure* in all cases of pulmonary tuberculosis or suspected pulmonary tuberculosis.

The commonest overt indication of laryngeal invasion is given in the voice. Hoarseness, at first perhaps slight, gradually becomes progressively worse, the voice taking on a peculiar weak huskiness, which may not be much more in the later stages than a rough whisper—a characteristic sound when combined, as it often is, with a rattling, incomplete cough.

When the stage of ulceration is reached, especially when the ulcerated surfaces lie where they are moved or irritated in the act of deglutition, then the patient may become the victim of the ominous and distressing odynphagia—pain on swallowing—which, by its interference with the taking of food, renders his downward progress precipitate. This suffering is often intensified by dysphagia from swelling of the epiglottis or arytenoids, by reason of which fluids enter the larynx during attempts at deglutition and excite distressing spasm and choking.

The appearances in the larynx *on examination* show great variety.

In the earliest stages nothing may be visible but the signs of catarrhal laryngitis;—congestion of the mucosa, the cords particularly manifesting abnormal redness. This redness and congestion is frequently of a patchy and irregular distribution, and if the background, as it were, of mucous membrane is pale and anæmic, if, besides, the pharyngeal wall shows an unusual pallor, while the lips and other mucous surfaces show normal redness, then the case should be regarded with grave suspicion, even although no evidence of any infiltration can be discovered. At this stage, a myasthenic weakness or absence of the voice simulating hysterical aphonia is not unusual.

In the stage of deposition or infiltration, the arytenoid, and particularly the inter-arytenoid regions are usually the first to be selected by the disease. The arytenoids, one or both, become swollen, rounded or pear-shaped, and semi-translucent, not, it is said, from true œdema, although the swelling looks like œdema, but from tuberculous infiltration and inflammation. It differs from simple inflammatory œdema in producing less bulky swelling, while it is, of course, much more chronic.

In the inter-arytenoid region the tuberculous swelling produces an appearance closely resembling the protuberant outgrowths of syphilitic or simple pachydermia. But the tuberculous lesion is more pale. In this region ulcers readily form, and thin granulations projecting from the inter-arytenoid region frequently conceal the ulcer, so that on examination the presence of the latter may be entirely overlooked, unless a direct laryngoscopic examination is made.

Along with, or independent of these changes, one or both of the ary-epiglottic folds and the adjoining ventricular bands may show swelling and ulceration.

The vocal cords frequently remain of a normal colour and shape after the rest of the larynx has been invaded by the disease, but sooner or later their involvement occurs, and indeed the disease may attack the cords and their neighbourhood before the arytenoid region. Infiltration changes their flat, even contour into a full and " soda-water-bottle shape" (St. Clair Thomson), while ulceration, superficial, irregular, and greyish on the surface, appears; at first the ulcers are small and discrete, but they tend to coalesce. Here also sprouting granulations spring up, and with the surrounding infiltration, lead to an entire alteration in the appearance of the glottic isthmus and walls.

The ventricular bands, when affected with tubercle, undergo tumefaction, and conceal the vocal cords. The epiglottis becomes thick and swollen, and when puffed up with pseudo-œdema assumes the classical " turban " or sausage shape. The destructive action of ulceration is also early seen in the epiglottis, and may lead to its destruction, only a slight stump remaining. Ulcers on the epiglottis, and on the pharyngeal side of the arytenoids, being exposed to the friction of food passing over them, are a common source of the agonizing odynphagia of the latter stages of laryngeal tuberculosis.

In the subglottic region the infiltration and granulations sprouting into the free lumen lead to narrowing of the airway, and appear on inspection as swellings below the vocal cords.

Perichondritis may set in relatively early in the course of the disease, but it is most frequent in the old cases. This complication is probably due to a secondary septic infection of the tubercle-riddled tissues, and it causes a massive necrosis of the cartilage attacked—most commonly the arytenoid. Abscess forms and breaks with a thin purulent discharge, which proceeds through one or more sinuses or openings usually into the interior of the larynx, although when the thyroid cartilage is the seat of perichondritis, the abscess may form on its outer aspect, and necessitate opening from the neck.

The symptoms of perichondritis and its effects have already been described (see p. 109), and those due to tuberculous perichondritis resemble the others in the general features. The occurrence of perichondritis in tuberculosis gravely compromises the patient's chances.

The foregoing account gives a sketch of the more frequent types of laryngeal tuberculosis, but there is no laryngeal disease which presents so many varieties. Among others, the following may be mentioned.

First, tuberculosis may remain more or less localized to one area, the epiglottis or the inter-arytenoid region for example, manifesting an indolence and a tendency to fibrosis that may, as a matter of fact, slowly end in recovery. More often, however, the disease becomes generalized and proves to be incurable.

Another rare variety is that where the *tuberculoma* forms. This is a well-defined, smooth, pale, rounded tumour, which, it is sometimes said, rarely ulcerates, and is not associated with tuberculosis elsewhere in the larynx. Most authorities agree, however, that this form of the disease generally assumes, sooner or later, the aspect of typical laryngeal tuberculosis.

Finally, mention must be made of a variety which, although by no means rare, does not always receive the emphasis worthy of its gloomy significance. To this type we have already alluded in an earlier section under the description of *general grey infiltration*. The whole of the larynx seems to be uniformly and equally involved, and the mucous surface shows a finely granular appearance, the arytenoids, ary-epiglottic folds and epiglottis being equally affected, and in the grey pallor such ulceration as is present is difficult to see and to define. So little, indeed, does the colour of the ulcerated surface differ from that of the still intact mucous membrane, that it is only by the obvious erosion and crumbled appearance of the outlines that we are aware that ulceration has taken place. This is particularly true of the ventricular bands and the vocal cords, the movements of which are stiff and imperfect from the infiltration and enfeeblement of their muscles. The only effect of their efforts is a toneless whisper and an imperfect cough which fails to eject from the laryngeal surface the stringy and tailing muco-purulent secretion that is hanging upon it.

General grey infiltration is the terminal stage of many of the varieties we have been describing, but it may appear right at the very beginning of tuberculous disease of the larynx.

Diagnosis.—There is no disease of the larynx that is not at times simulated by tuberculosis.

What looks like simple catarrh may prove to be the first indication of the graver disease, and catarrhal laryngitis occurring in a phthisical patient should be regarded with misgiving unless it promptly and permanently disappears.

In males over middle life, a tuberculous deposit in and about the ventricular band and vocal cord of one side may in its external appearance and in its immobilizing of the cord so closely resemble cancer as to deceive even the expert.

What look like the papilliform projections of simple pachydermia either on the cords or in the inter-arytenoid region may turn out to be tuberculosis, and even apparently simple neoplasms of the vocal cord like fibromata or papillomata may also prove to be the heralds of laryngeal phthisis.

Syphilis, likewise, may be simulated when the tuberculous disease takes on a general fibrotic tendency, but perhaps syphilis is less often mistaken for tuberculosis than the other diseases we have mentioned. Allusion to mixed syphilitic and tuberculous disease has already been made.

In all these mimicries, however, the false appearance is only temporary. Sooner or later the real nature of the disease declares itself unmistakably.

The points to notice are: the pallid colour of the infiltrated areas, and the tendency for the arytenoid eminences to become swollen and rounded.

Doubt need scarcely arise when the pulmonary disease is already known to be present, and so the clinical rule holds good that the lungs should be examined by means of X-rays as well as by the stethoscope in all laryngeal disease, and if there is any expectoration it should be searched for the tubercle bacillus.

Where it is important to make a prompt diagnosis in a doubtful case, a portion of the diseased area should be removed and examined microscopically.

From lupus, a rare disease in the larynx (see p. 125), tuberculosis is distinguished by its irregular distribution, its production of pseudo-œdema, its tendency to avoid the epiglottis in the early stages, its moist surface, its tendency to infiltrate deeply and to cause perichondritis, the painful character of its ulcers, and its progressive course.

Prognosis.—Tuberculosis of the larynx is always serious, and as a complication of phthisis pulmonalis it frequently introduces the element into the situation that determines the issue. The danger consists in its resistance to treatment, and in its tendency to interfere with nutrition by inducing odynphagia.

Of the several varieties of the disease, those types which manifest infiltration, especially if only regional, without ulceration or with only slight ulceration, are the least unfavourable, while the worst type is the general grey infiltration.

A relatively favourable variety is that rare form in which the disease is confined to the epiglottis, as it may then be possible to effect its eradication surgically.

The presence of ulceration, introducing as it does the factor of secondary infections, always renders the prognosis grave.

The chances in mixed syphilis and tuberculosis are very poor.

As in the case of the lungs so with the larynx, the prognosis chiefly depends upon the response to treatment. It is often (but not invariably) found that lungs and larynx progress either favourably or unfavourably in harmony with each other.

Sir St. Clair Thomson points out that cases which come to throat clinics do badly because the disease is advanced before such patients recognize its presence. On the other hand, cases in sanatorium where the laryngeal disease is diagnosed early as a result of laryngeal examination have a much better chance of recovery.

Treatment.—General treatment is all-important, as the disease is one primarily of pulmonary tuberculosis. Thus, the first question to be decided is whether the case is one suitable for sanatorium treatment. The condition of the larynx must be considered in determining this question, but it may be said that if the state of the lungs and the general condition of the patient favour the adoption of the sanatorium treatment, fairly advanced laryngeal disease should not be considered as a contra-indication. The only laryngeal contra-indications of sanatorium treatment are intractable odynphagia preventing adequate nutrition, and the general grey infiltration with ulceration. The reason for this is, that remarkable improvement, even amounting to cure of the laryngeal disease, may result from sanatorium treatment when the general condition of the patient responds favourably to it.

Locally, rest should be insisted upon, even in the earliest cases. That is to say, the patient should preserve ***absolute silence***, communicating only by writing. This should be maintained, other factors permitting, for six months. At the end of that period, the rule may be relaxed.

For the soothing of inflamed areas, simple oily laryngeal sprays with menthol, chloretone, and so on, may be employed:

℞.	Menthol,	grs. x.
	Paraffin Liq.,	ad. ℥ i.

℞.	Chloretone,	gr. v.
	Menthol,	gr. x.
	Paraffin Liq.,	ad. ℥ i.

Caustics, astringents, and irritants generally are to be condemned, but the constant wearing of an inhaler is decidedly useful, especially for the soothing of cough and laryngeal tickling and irritation.

For this may be used

℞. Creosot.
Spt. Chlorof.
Spt. Vin. Rect. equal parts,
Sig.—A few drops on the sponge of the inhaler.

When it can be easily accomplished, the intratracheal injection of 10 per cent. menthol in olive oil, introduced by Walker Downie of Glasgow, has proved useful in my hands, and I have even been able to teach patients to administer it to themselves.

Otherwise, all laryngeal swabbings and applications should be strictly eschewed.

Curetting and other surgical measures, though warmly advocated at one time, should be confined to the removal of granulations and outgrowths, *when these are blocking the airway.* To this there is an exception, and that is in the case of tuberculosis limited to the epiglottis; in which case the epiglottis may be removed by Lake's epiglottis punch. (Fig. 26.) The loss of this appendage to the larynx has no effect upon the act of swallowing.

The other laryngeal manipulations, such as curetting, may be conveniently performed with suspension laryngoscopy.—Care should be taken not to carry the operation too deep, as the wounds are apt to bleed severely, and these patients are ill-adapted to withstand such a mishap.

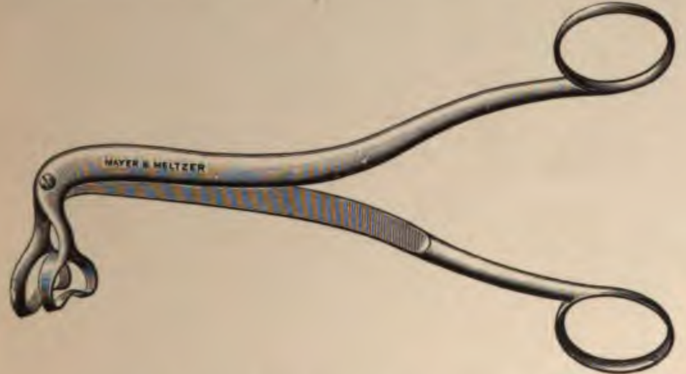

FIG. 26.—Lake's Epiglottis Punch Forceps.

Of all methods of local treatment, none is reputed to be so productive of benefit in suitable cases as *galvano-cautery puncture.* Under cocaine, and either by direct or indirect laryngoscopy, the terminals here figured (Fig. 27) are brought to a white heat, and plunged deep into infiltrated and even into ulcerated areas. Several punctures are made at one sitting, the *séance* being repeated monthly or thereabouts. There is very little reaction induced and so no great œdema, but care should be taken not to plunge the heated cautery point as deep as the underlying cartilage.

The galvano-cautery puncture is said to be a useful adjunct to the sanatorium and silence treatment, but it should not be used in the presence of active pulmonary and general symptoms.

My personal experience of this treatment is, as a matter of fact, not favourable.

In cases where dyspnœa is present as a result of laryngeal obstruction by the disease, *tracheotomy* may be required as an urgent measure, but its influence upon the pulmonary disease is not good.

In the terminal stages of laryngeal tuberculosis where the patient's life is made miserable by odynphagia, the distress of swallowing may be mitigated by the insufflation or by the inspiring through Leduc's tube of 3 to 5 grains of orthoform or anæsthesin about an hour before the principal meal of the day.

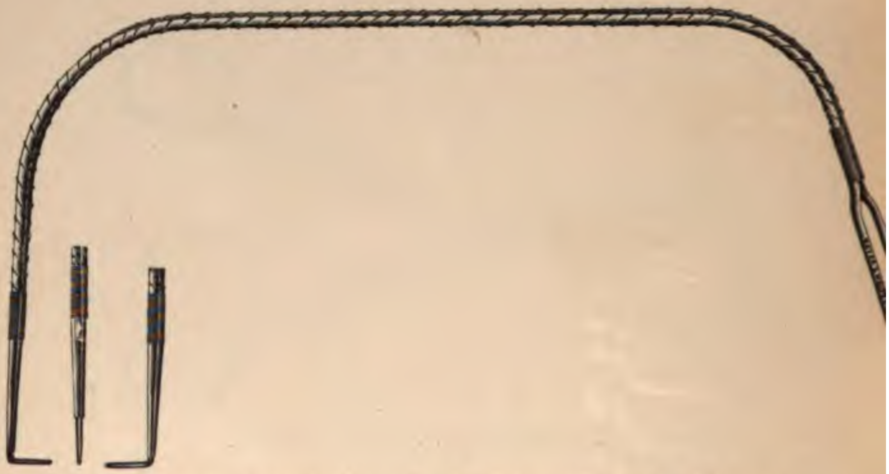

FIG. 27.—Author's Electrodes for Galvano-Puncture of Larynx.

It is also sometimes of value for the relief of painful deglutition to make *an injection of alcohol into the trunk of the superior laryngeal nerve.* The needle is entered so as to strike the greater cornu of the hyoid one inch behind the lesser cornu. Its point is then depressed, so that it hitches against the lower border of the greater cornu. Here the needle is in contact with the thyro-hyoid membrane, and it is now directed downwards and forwards inside the thyroid cartilage, and while it is being pushed in this direction, the piston is slowly closed, and the alcohol injected along the course of the nerve. (After Courtenay Yorke, quoted by Sir St. Clair Thomson.)

When swallowing is painful and difficult, we must, naturally, give the patient only soft solids or liquids to swallow, but in this matter, patients may be left to choose for themselves. In like manner also they soon discover what attitudes to adopt to facilitate the passage of the food, the most serviceable perhaps being that of Wolfenden, in which the patient lies face downwards on a couch, with his head over the end of it, and sucks through a tube the nourishment from a vessel placed below him on the floor.

LUPUS OF THE LARYNX

Lupus is rarely seen in the larynx, and never, it is said, without evidences of the disease in the nose or pharynx.

It occurs oftenest on the epiglottis, which is frequently destroyed by the disease. The deposit shows pale, dry-looking lupus nodules breaking down to form a dry, shallow ulcer, with little or no reactive redness about it. Other deposits may be seated elsewhere in the larynx, but it seldom attacks the cords.

Diagnosis.—It is essential not to confuse lupus with tuberculosis.

The *prognosis* of the disease is generally said to be favourable, but the opinion has been expressed that lupus in the larynx often undergoes a conversion into the more active forms of tuberculosis, and ends unfavourably.

Treatment.—In addition to general constitutional treatment by good feeding, fresh air, and tonics, arsenic being specially recommended, the galvano-cautery puncture of the lupoid deposits in the larynx should be carried out.

Lupoid deposits elsewhere must be attended to (see pp. 76 and 291), and the possibility of tuberculosis in other organs, especially in the lungs, should be borne in mind.

BENIGN NEOPLASMS OF THE LARYNX

Papilloma is the commonest variety of innocent new growth. It occurs chiefly in children and young adults. In structure, the papilloma is a warty growth, consisting of a core of connective tissue with blood-vessels, and an epithelial covering. Generally multiple, papillomata vary in size from a millet-seed to a walnut. They are pale pink in colour, and present a mulberry or cauliflower-looking surface. (Figs. 28 and 29.)

They are usually pedunculated, firm, elastic, and not liable to bleed.

Papillomata most frequently grow from the free border of the vocal cords, affecting the middle or anterior half, or the anterior commissure. When multiple, they are often scattered about in clumps elsewhere in the larynx. Indeed they may extend into the pharynx and down into the trachea, and they have even been known to grow on the granulation tissue of a tracheotomy opening. (Dundas Grant). When papillomata manifest growth so luxuriant as this they should be regarded as locally infective, if not locally "malignant," and as dangerous to life, since asphyxia may be induced by the impediment they offer to the passage of the air through the glottis. This is specially remarked in children.

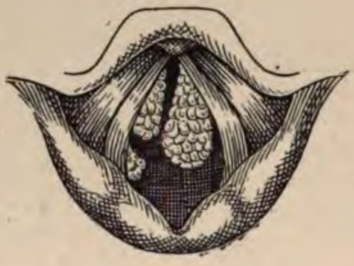

FIG. 28.—Papillomata of the Larynx (Andrew Wylie).

Diagnosis.—A papillomatous clump on one vocal cord in an adult over middle age should be suspected, as epithelioma in its early stages may present a papilloma-like surface. The malignant growth, however, is accompanied with infiltration of the tissue around it. All laryngeal growths, when removed, should be examined by a competent microscopist.

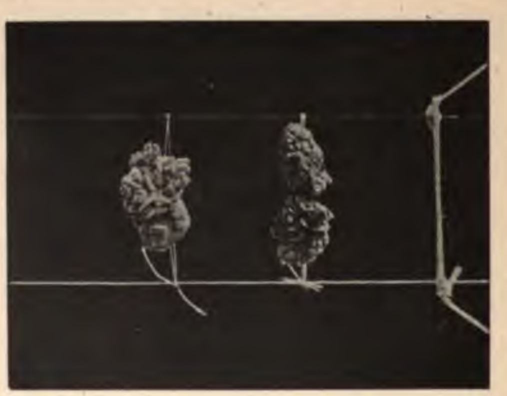

FIG. 29.—Large Papillomatous Growth removed from Larynx (Andrew Wylie).

In children, persistent hoarseness, or stridor, whether continuous or recurrent, should lead to an examination for papillomata. Children can be trained to submit cheerfully to examination with the laryngeal mirror.

Prognosis.—In view of the strong tendency to recurrence, and occasionally to prolific growth, the prognosis should be guarded. Nevertheless, when papillomata are thoroughly removed, their recurrence is not common. The difficulty, however, lies in their thorough removal.

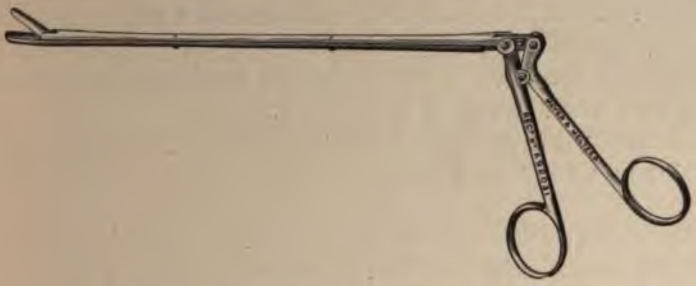

FIG. 30.—Paterson's Direct Laryngeal Forceps.

Treatment.—*In Adults*.—By *indirect laryngoscopy under cocaine*, and using the laryngeal forceps of Morell Mackenzie or Dundas Grant (Fig. 33), the growths are seized and evulsed, (Fig. 29), each clump being attacked and systematically eradicated. Several *séances* may be required to clear out all the colonies.

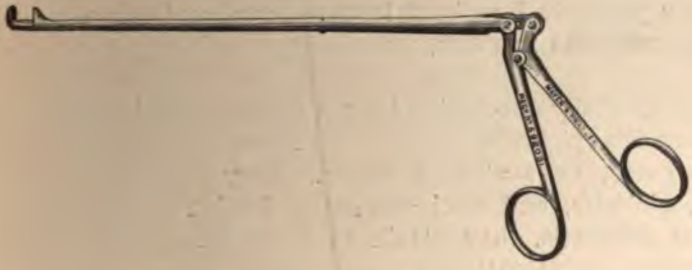

FIG. 31.—The Author's Direct Laryngeal Forceps.

The same may be performed under *direct laryngoscopy*, using Paterson's or the author's direct forceps. (Figs. 30 and 31.) After the operation, the bases of the growths may be touched with lactic acid.

In *children* laryngeal papillomata are most successfully dealt with by removal under suspension laryngoscopy. Chloroform is necessary, and after the apparatus has been adjusted, the growths are removed by suitably long forceps, and their bases are then curetted.

In these manipulations, it is advisable to handle each growth as if it were capable of disseminating its species by contact. As soon as the papilloma is seized, therefore, it is removed in such a way as to avoid contact with the mucous membrane of the larynx or pharynx.

A low tracheotomy is often a necessary antecedent of this operation in children, and if there has been a recurrence of the growths, the child should wear the tube for a considerable period, if necessary for several years, as the tendency to recurrence disappears when the tube is retained.

Internally, the prolonged administration of arsenic or iodide of potassium may be tried.

Fibromata, which are not uncommon tumours in the larynx of adults, are usually found on the upper surface of the anterior half of one or other vocal cord. Wyatt Wingrave has shown that they are frequently the product of the organization of localized submucous hæmorrhage. Their structure is of fibrous tissue, frequently œdematous, and shows traces of their hæmatogenous origin.

This neoplasm is generally solitary, sessile, light pink or red, and smooth. It is, however, occasionally stalked, and may further resemble a papilloma in being granular on the surface.

Its removal may be effected under cocaine, either by indirect or very precisely by direct laryngoscopy, and when removed, it does not tend to recur.

Cysts, usually developing from a mucous gland, are found at times on the epiglottis, and more rarely on the ventricular bands, vocal cords, or arytenoid region. They appear as smooth, tense, globular, and semi-translucent bodies, which, especially on the epiglottis, may attain to a considerable size without attracting any attention.

Treatment.—Puncture or incision of the cyst being insufficient, a portion of the wall should be excised. Even then there may be a recurrence.

Other neoplasms, such as angeioma, myxoma, lipoma, chondroma, and adenoma are rarely found in the larynx.

The *Symptoms of benign neoplasm* in the larynx (other than papilloma) naturally depend upon the position and size of the tumour. Quite a small growth, if it is on the edge of the cord, will attract early attention, by inducing hoarseness, while, on the other hand, a tumour on the ventricular bands, for example, will grow to quite a large size before the patient is aware of anything amiss, and then the first symptoms to appear may be stridor and dyspnœa.

On examination, the cause of the symptoms is, as a rule, easily detected, but small growths located about the anterior commissure readily escape a casual inspection.

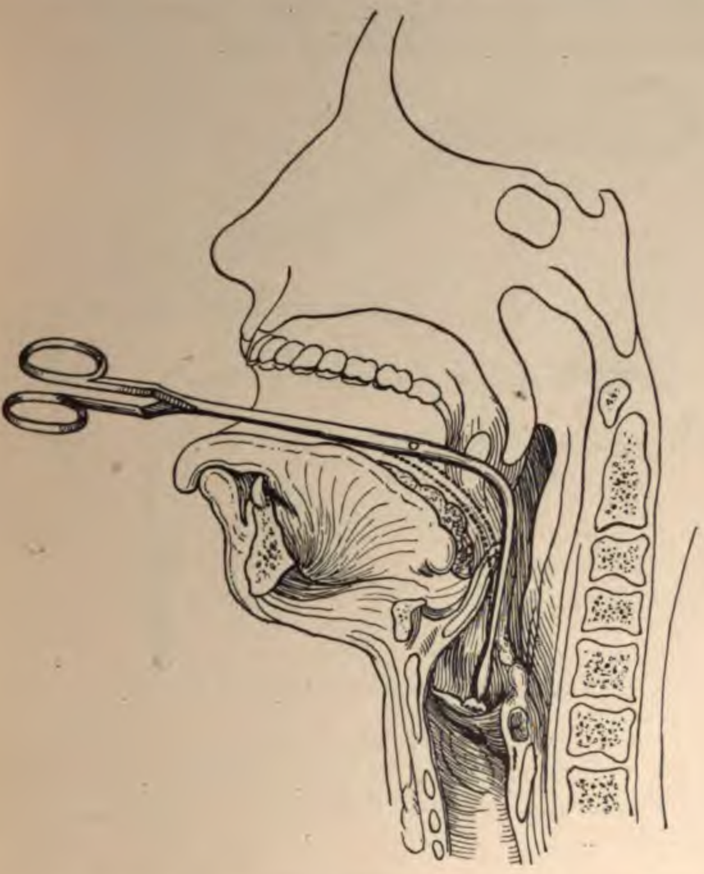

FIG. 32.—Removal of a Neoplasm from the Vocal Cord by the Indirect Method (from " Diseases of the Throat and Nose " by Morell Mackenzie).

Treatment.—The removal of small, solitary laryngeal neoplasms without injury to the cord is a little operation necessitating skilled and delicate manipulation, whether undertaken by the direct or by the indirect method. Efficient local anæsthetization is essential (see p. 92), and the patient should be trained to co-operate with the surgeon, in fixing and moving the head to command. (Fig. 32.)

For tumours on the edge of the cords, Dundas Grant's laryngeal forceps (Fig. 33) are useful, while Morell Mackenzie's forceps are probably the most popular for tumours situated elsewhere.

Operating by the direct method has the merit of being more simple and hence more precise, but the indirect method makes, on the whole, less demand upon the patient's fortitude.

After the larynx has been cleared, it should be rested for several days by the patient keeping silent.

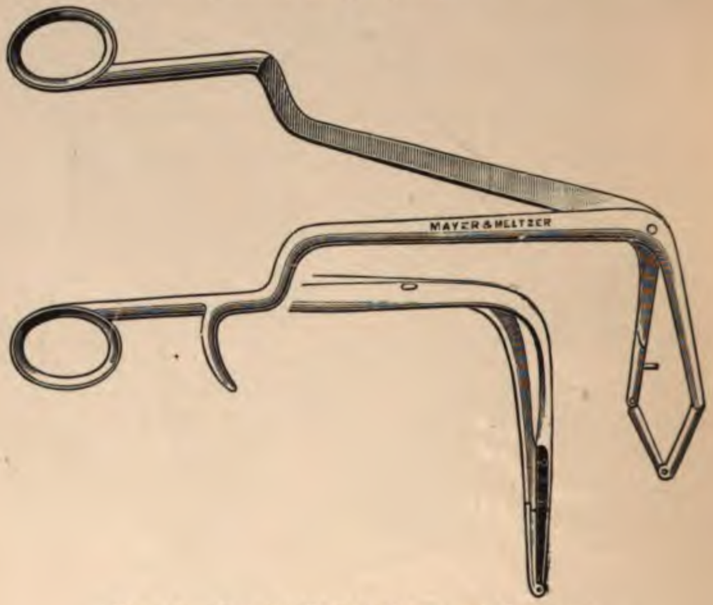

Fig. 33.—Dundas Grant's Laryngeal Forceps.

These last varieties of neoplasm, unlike the papillomata and the malignant group, do not readily recur. If, therefore, recurrence does take place the diagnosis should be carefully re-considered.

MALIGNANT NEOPLASMS OF THE LARYNX—CANCER OF THE LARYNX

Sarcoma of the larynx is very rare.

Carcinoma.—The common type of cancer of the larynx is epithelioma. It occurs either as a primary growth or by extension from neighbouring parts, almost never by metastasis.

The factors which predispose to the cancerous process in the larynx are not yet clearly determined. It is commoner in males than in females, and, like cancer elsewhere, it is commoner after than before the age of forty. Nevertheless it does not spare women and it has been observed before middle life not infrequently.

Chronic laryngitis, laryngeal syphilis, and other forms of local irritation probably favour the onset of the disease, but it has not yet been definitely proved that they do so.

Clinically, three varieties of laryngeal cancer are distinguished according to their site of origin:

(1) *Intrinsic Cancer* when the growth is limited to the vocal cords, the ventricular bands, the ventricles, or the sub-glottic region as far as the upper border of the cricoid cartilage.

(2) *Extrinsic Cancer*, when the epiglottis, the arytenoid region, the ary-epiglottic folds (the epi-laryngeal region), or pyriform sinuses are attacked; and,

(3) *Mixed* cases, where areas coming under both of the former are implicated.

From the point of view of prognosis and treatment, these varieties differ profoundly from each other, for the following reasons: The lymphatic system of the larynx is, as it were, self-contained, for although the laryngeal lymphatic vessels anastomose freely with each other, it is only through a long and circuitous route that they communicate with the lymphatics of neighbouring tissues; for this reason, cancer of the area of the laryngeal lymphatic system remains localized for a long time before it infects the cervical lymphatic glands. This area is the area of *intrinsic* cancer. The regions where *extrinsic* cancer forms, however, are provided with lymphatics which do communicate freely with neighbouring lymphatics and lymphatic glands; consequently, cancer affecting those areas tends to become widespread more rapidly. Hence it is of great importance to be able to note the exact site and extent of the growth.

The glands which become enlarged in supraglottic cancer, whether intrinsic or extrinsic, are those found at the level of the upper border of the thyroid cartilage, between the larynx and carotid vessels. In infraglottic cancer, the glands in the middle line in front of the cricoid and trachea, or those in the carotid chain on a level with the cricoid, are the most liable to infection.

Another reason why intrinsic cancer is the less dangerous is that its rate of local growth is slower than that of extrinsic cancer. Finally, while it remains limited to the interior of the larynx, it is much more favourably situated for complete removal than is the extrinsic disease.

Symptoms.—The early stages of laryngeal cancer may pass without giving any warning of the presence of the disease, as, for example, when the growth begins on an ary-epiglottic fold. But when the cord is the first seat of the disease, hoarseness sets in as an early symptom, and it is this generally which brings the patient to the doctor. The amount of hoarseness is often surprisingly great in comparison with the extent of the growth, but the symptom is produced not so much by the bulk of the new growth as by an infiltration which quickly leads to interference with the mobility of the cord.

In intrinsic cases, as the disease progresses, the hoarseness becomes more pronounced until only a rough whisper is audible.

In extrinsic cases, the first symptom to attract notice is generally difficulty and pain on swallowing. But, as we have said, the disease is often far advanced before any signs arise, and it is by no means unusual to find that the first symptom noticed by the patient is the presence of enlarged glands in the neck.

Both types tend, as the bulk of the growth increases, to interfere more and more seriously with respiration so that in inoperable cases tracheotomy, as a measure of relief, is almost always required

In the final stages, the pain on swallowing becomes very severe; the saliva is allowed to dribble from the mouth; the breath is fœtid; until emaciation, hæmorrhage, cachexia, and progressive misery and weakness, or the occurrence of septic pneumonia lead to death.

Examination.—Cancer of the larynx assumes many forms in its earlier appearances, but it presents also certain characters common to all, and these are generally sufficient to awaken suspicion and sometimes to establish the diagnosis beyond doubt.

On the vocal cord in its earliest stages it most frequently appears either as a white or greyish warty growth on one cord, resembling papilloma; or as a diffuse ill-defined growth on the surface of the cord, infiltrating and thickening its substance; or else it appears as an uneven reddish fringe along the edge of the cord.

Elsewhere in the larynx epithelioma shows as a deep-pink infiltration with a rough, uneven surface. On the epiglottis, as might be expected, the colour is lighter—greyish or pink—and here ulceration begins early.

At first the progress of the disease is slow, and in the vocal cords it may seem for prolonged periods to be almost stationary, but after ulceration has taken place, the disease extends more rapidly, spreading, in the case of the cords, backwards to the inter-arytenoid region, or forward to the anterior commissure, and thence to the other cord, so that the glottis may be encircled by the disease. Thence it spreads up into the ventricular bands, and down, but more slowly, into the subglottic region, until, it may be, the whole of the larynx becomes filled with the sprouting growth. (See Fig. 37.)

When the extrinsic region is affected, the route taken by the infiltration is upwards towards the pharynx, or outwards on to the pharyngeal wall of the larynx. (For " post-cricoid cancer," see p. 66.)

The epitheliomatous ulcer presents an irregular, greyish or whitish floor. It bleeds readily and it is very hard and tender to the touch. As Stuart Low and Wilfred Trotter have pointed out, it is possible not infrequently to palpate laryngeal growths with the forefinger inserted through the mouth, and as the information thus obtained is of great value, the attempt should always be made.

In its early stages on the cord, the cancerous growth often gives the impression of deep infiltration by reason of the thickness and reddish colour of the mucosa around it. The diseased surface has a softish appearance, while a congestion of the affected cord as compared with the other cord is always a suspicious sign. Considerable emphasis is usually laid upon the fact that a cord affected with cancer manifests on movement a heaviness or sluggishness as if it were loaded with some burden, and this is certainly a phenomenon to be noted. But it is not limited to cancer. One finds it also in unilateral tuberculosis when it affects the ventricular band. Finally, however, the infiltration and ulceration of cancer tend to immobilize the cord altogether.

In the later stages, the appearances, if the case has been watched, become unmistakable. The foul, fungating ulceration, the immobility of the cord, and the stony hard cervical glands leave no doubt as to the nature of the disease. But even in its later stages, the real disease may be masked by septic perichondritis, and this, in point of fact, may be the first event which leads the patient to seek relief, so that any laryngeal inflammation with external tenderness should always be closely watched for future developments.

Diagnosis.—Hoarseness lasting longer than a fortnight, at any age, should lead to a laryngoscopic examination, with the thought of papilloma in children; of tuberculosis in early adult life; of cancer in later life.

Cancer of the larynx may be mistaken for (a) simple chronic laryngitis; (b) syphilis; (c) tuberculosis, or (d) a benign tumour.

In *chronic laryngitis*, pachydermia affecting the cords comes to resemble cancer if the pachydermatous thickening affects one cord more than the other. The question then arises whether epithelioma is supervening upon the chronic laryngitis. In that case, the likelihood of epithelioma will be strengthened if the suspected cord shows a lagging movement as compared with its neighbour, or if an ulcer develops on the edge or surface of the cord and persists. The irregularities of the pachydermatous cord, it must be confessed, are themselves often hard to distinguish from an ulcer.

Syphilis in its several forms may be diagnosed from the comparatively short history, from the existence of signs of syphilis elsewhere, from the preference manifested by the disease for parts other than the cords, and from the result of anti-syphilitic treatment. But it should be remembered that in the larynx, just as elsewhere, a certain amount of **improvement after the administration of potassium iodide should not be regarded as definitely excluding cancer.**

A syphilitic perichondritis may be indistinguishable from cancerous perichondritis.

With regard to the Wassermann reaction, we must remember that a positive Wassermann only indicates that the patient is a syphilitic; it does not decide the nature of any particular lesion.

From *tubercle*, cancer is distinguished by the preference of the latter for the cords; by the patient being, as a rule, over middle age; by the absence of disease in the lungs, and of tubercle bacilli in the sputum. In addition, the tubercular ulcer in the early stages is shallow, pale, and shows no infiltration around the edges. At the same time cases come for diagnosis in which it is not easy to come to a decision, as we have already seen. (See p. 120.) In such puzzling circumstances the most reliable test is time. In tuberculosis, the picture changes more rapidly than in early cancer, and the changes bring more and more into prominence the formerly latent tuberculous characteristics.

Innocent tumours of the larynx are of slow growth, show no tendency to infiltrate the surrounding structures, do not ulcerate, and do not interfere, save mechanically by their bulk, with the mobility of the affected cord. Benign tumours on the cords also are most common on the anterior half, while cancer is commonest on the posterior third of the cords.

Papilloma in its tendency to recurrence and to diffusion is the benign growth which most closely resembles cancer, and papillomatous neoplasms over the age of forty years always arouse apprehension. But the appearance of a second or third papillomatous cluster some distance from the first, would tend to relieve anxiety. Nevertheless, as we have already remarked, all apparently benign neoplasms of the larynx should, after removal, be handed to a competent microscopist for examination. And indeed in all cases of doubt, a portion of the diseased tissue should be removed for microscopic examination.

This little operation can be undertaken under local anæsthesia, and it is important to obtain the specimen from that part of the diseased area which shows the greatest change, and to include in the portion removed all the layers of the tissue superficial and deep. That being so, the writer advises the direct method as enabling the operator to be more precise in his manipulation. Punch forceps of a sensibly large pattern should be employed, otherwise the surface only will be nibbled.

If the microscopist's report is glaringly contrary to what is suggested clinically, a second, or even a third specimen should be obtained and examined, and the same is true when the report is uncertain in tone. In this, also, as in many other clinical "tests," a negative response is of less value than a positive.

Finally, perhaps the most important of all factors in diagnosis is the effect of time upon the laryngeal appearances. In any doubtful case, short of urgency, the larynx may be inspected from week to week, or, if the progress is slow, at even longer intervals, while in the meantime the patient may be receiving anti-syphilitic remedies. The changes effected by time are frequently illuminating.

As has already been noted, the occurrence of perichondritis may be the first sign of cancer of the larynx, and in that case, so long as the acute inflammation is in existence, the underlying malignant disease may be entirely masked.

The *Treatment of Cancer of the Larynx* is radical or palliative according to the site and extent of the disease. The radical treatment consists in operative removal, by (a) thyrotomy (laryngo-fissure); (b) total laryngectomy; (c) subhyoid and transhyoid pharyngotomy.

THYROTOMY (LARYNGO-FISSURE)

(See Irwin Moore's articles in *Journ. of Laryng.*, vol. xxxiii).

Description.—The thyroid cartilage is opened through an incision in the middle line and the growth excised through the opening.

Indications.—Intrinsic cancer of larynx limited to one side in which the cartilage is not involved; the most favourable cancers are those limited to the cord, but the operation has also proved occasionally successful even when the disease has spread round the commissure to invade the other cord.

(Laryngo-fissure is also sometimes resorted to for the removal of foreign bodies impacted in the larynx when they cannot be removed *per vias naturales*; for cicatricial stenosis; sometimes for the removal of papillomata and of other benign neoplasms.)

Contra-Indications.—*Local.*—The operation is unsuited for cancer which has grown beyond the "intrinsic" limits, and for cancer which affects both sides of the larynx, unless perhaps when the growth has passed over to the other side by the anterior commissure, and only to a very slight extent.

It is also unsuited to cases in which the growth has penetrated the thyroid cartilage, an extension which may be detected by palpation of the thyroid ala from without, previous to operation.

General.—Disease of the lungs, heart or kidneys. Experience proves, however, that senility, if fairly robust, is no contra-indication.

Operation.—As in all operations on the larynx, pharynx, or nose, the mouth is cleansed previous to operating, by the removal of all carious teeth, and the administration of an antiseptic mouth-wash.

On the operating table the patient lies on his back, with a small pillow under the shoulders, to extend the neck, and render prominent the larynx and trachea. General anæsthesia by chloroform is employed.

An incision is made in the middle line of the neck from the level of the hyoid bone to the upper edge of the sternum, and the soft parts are divided in this line until the thyroid and cricoid cartilages, the trachea and the isthmus of the thyroid body are exposed and clearly defined. The thyroid isthmus is divided in the middle line to expose the underlying trachea, and after injecting a few drops of a 10 per cent. solution of cocaine into its lumen (see p. 181), the trachea is opened and a tracheotomy tube inserted. The anæsthetic is now administered through the tracheotomy tube.

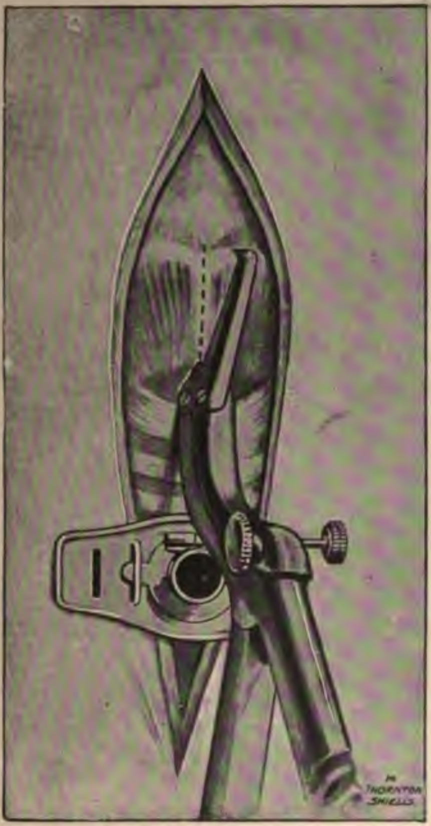

FIG. 34.—Shears cutting through the Thyroid Cartilage. One blade has been passed through the crico-thyroid membrane into the interior of the larynx (from Irwin Moore).

Next, the thyroid and cricoid cartilages having been cleared in the middle line, the former is divided from its lower border to its notch by means of thyroid shears (Fig. 34), or, if the cartilage is ossified, by means of Irwin Moore's thyroid saw.

The alæ of the thyroid cartilage are now separated and held apart by retractors so as to expose the interior of the larynx. Some of the mucous membrane of the larynx lining the thyroid cartilage may require snipping, and one has sometimes to divide partially the thyro-hyoid membrane in the middle line. A sponge is now packed up towards the pharynx to prevent contamination from the mouth discharges, and another small sponge attached to a string is passed through the laryngeal opening down into the trachea as far as the tracheotomy tube in order to intercept any blood that may flow down, and the laryngeal cavity is packed for a few minutes with gauze soaked in 5 per cent. solution of cocaine in solution of adrenalin (1-1000).

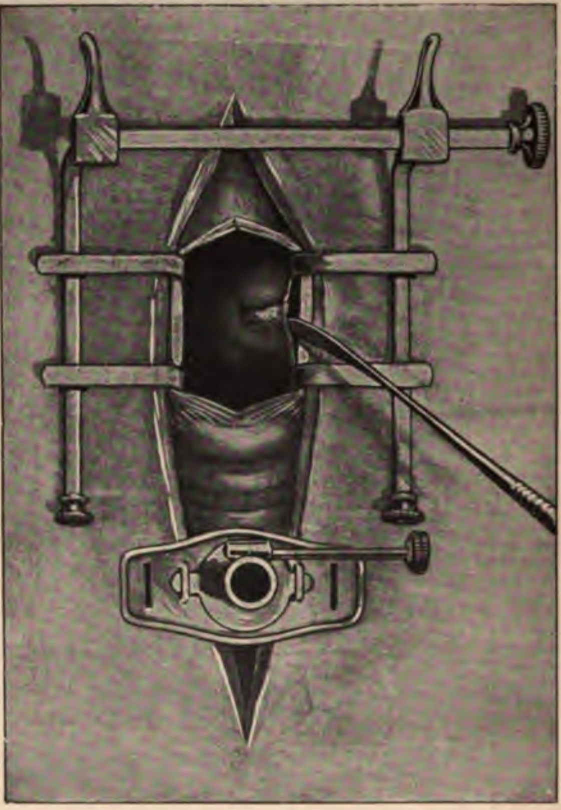

FIG. 35.—Larynx opened and elevator passing under the perichondrium to separate the growth-bearing tissue from the thyroid cartilage (Irwin Moore).

This having been removed, the infiltration of the growth will show up as a dull red area against the surrounding anæmic laryngeal mucosa; the growth always proves to be more extensive than it appeared on laryngoscopy.

A blunt periosteum elevator is then insinuated into the cut edge of the laryngeal mucosa of the affected side between the thyroid ala and its perichondrium (Fig. 35), and working backwards and up and down, we separate the soft tissues bearing the growth in an entire mass from the cartilage. With curved scissors the separation of the growth is completed by successive snips carried through the elevated mucosa, above, below and beyond the diseased area, the division passing through healthy tissue about half an inch clear of the growth all round. (Fig. 36.) Care is taken to avoid touching the area of actual disease, as that might contaminate the rest of the wound. The excision, indeed,

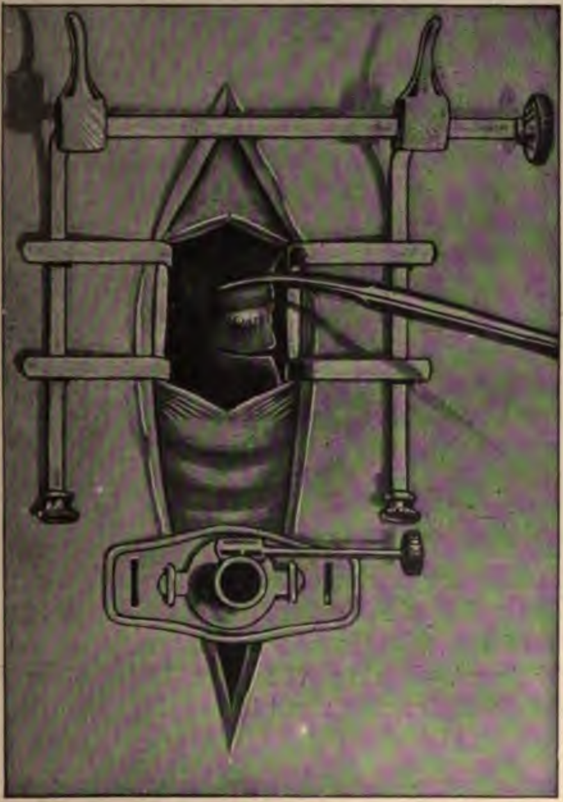

FIG. 36.—The growth-bearing tissue having been separated from the cartilage, it is being isolated by scissors (Irwin Moore).

may be most safely accomplished with the diathermy knife, care being taken not to touch the cartilage. (See p. 65.)

In effecting this removal, the arytenoid region should if possible be spared. If it must be included, the surgeon must be prepared for smart hæmorrhage, and also for some difficulty in deglutition during convalescence. The former can be stopped by pressure forceps and ligatures; the latter avoided by feeding the patient through a nasal tube for ten days.

After removal of the mass, a small vessel in the neighbourhood of the arytenoid is liable to bleed and should be ligatured. For the rest, as in all neck operations, the patient is allowed to come round partially from the anæsthetic, and his neck is relaxed by flexing the head so as to reveal all bleeding points, in order that they may be ligatured. This completed, and the pharyngeal and tracheal sponges removed, the separated thyroid alæ are brought together by catgut sutures passed through the perichondrium The thyro-hyoid membrane, if it has been divided, is also sutured. Finally, the skin is sutured with silkworm gut, and a light dressing applied.

The tracheotomy tube should remain *in situ* for 24 hours, in case bleeding should occur while the patient is semi-conscious, as that may lead to death by asphyxia from the blood trickling into the lungs.

Difficulties.—The common difficulty is that of separating the divided thyroid alæ sufficiently to permit of satisfactory inspection and manipulation of the interior of the larynx. Relief may be obtained by incising the thyro-hyoid membrane or the upper half of the crico-thyroid membrane.

After-treatment.—During the first twenty-four hours the patient is watched for shock and hæmorrhage, but as soon as he is able, he should be propped up into the sitting position, as this eases his coughing and facilitates swallowing. The mouth should be kept clean by frequent washing-out, and it is a wise precaution to have all food and drink sterilized.

The danger is septic pneumonia from the entry of septic material into the bronchial system, but the danger can be avoided by strict attention to the above rules.

The patient, especially if old, may be allowed to get up out of bed as soon as he feels inclined.

Results.—According to figures collected by Sir St. Clair Thomson, the operation mortality of laryngo-fissure is about 6 per cent.; recurrence has been noted in about 22 per cent.; while 47 per cent. have remained free from recurrence for three years or longer. Thus, in suitable cases, and in expert hands, the operation mortality of laryngo-fissure is practically *nil*, and the mortality from recurrence of the epithelioma is as low as, or lower than, that in any other form of cancer, not excluding cancer of the lip.

The same authority warns us not to mistake a granuloma appearing in the anterior commissure shortly after the operation for a recurrence.

The result as regards the voice is generally remarkably good. The scar-tissue seems to gather up to form a new vocal cord, and the patient recovers a useful amount of phonation.

Recurrences are to be watched for, and may not appear for many years after the operation.

LARYNGECTOMY

Description.—After the trachea has been opened and shut off from the larynx at a preliminary operation, the whole larynx, including the cricoid, thyroid and arytenoid cartilages with their internal structures are removed in one piece. (See Fig. 37.)

Indications.—Cancer limited to the larynx, but exceeding the limits laid down as suitable for laryngo-fissure. (See p. 136.)

Contra-Indications.—Local.—Cancer which has extended and infiltrated tissues beyond the confines of the larynx, as up into the pharynx, or through the cartilages into the soft tissues of the neck.

General.—The operation is much more grave than laryngo-fissure, and should not be attempted unless the patient is otherwise healthy.

The operation mortality has been considerably reduced since the method was introduced of shutting off the trachea and bronchi from the laryngeal wound.

Preparation.—The cleansing of the mouth is of the first importance; carious teeth should be removed, and if there is any pyorrhœa, it is advisable to have all the teeth extracted. The major operation should not be undertaken until the dental wounds are entirely healed up. (See also p. 52.)

Operation.—There are two methods of dealing with the trachea.

First, a low tracheotomy is performed, and a tracheotomy tube is inserted. Then a fortnight later the major operation, the removal of the larynx, is performed.

Removal of the Larynx.—The anæsthetic is given through the tracheotomy tube. A vertical incision is made in the middle line from the hyoid bone to half an inch above the tracheotomy opening, a bridge of skin and soft tissues between the two wounds being left intact. A transverse incision is made at the upper end of the median incision along the lower border of the hyoid bone as far as the anterior border of the sterno-mastoid muscle on either side. A second transverse incision at the lower end extends likewise from the median incision to the anterior edge of the sterno-mastoids.

The median incision is now deepened until thyro-hyoid membrane, thyroid cartilage, crico-thyroid membrane, cricoid cartilage, and the upper two or three rings of the trachea are exposed.

The transverse incisions are deepened to the sterno-mastoid level through superficial layers of fascia and platysma.

A blunt elevator is now insinuated beneath the perichondrium over the thyroid cartilage in order to raise the perichondrium and the tissues over it from the thyroid alæ as far back as the pharyngeal constrictor on either side. This leads to the posterior edge of the thyroid cartilage. Next, with the elevator passed under the depressors of the hyoid, these muscles are cut through at the thyro-hyoid level, revealing the lateral portion of the thyro-hyoid membrane. At the lower end of the wound the muscles are raised off the cricoid, and the lobe of the thyroid gland is cleared and left. Some venous hæmorrhage at this spot is met with.

Drawing the half-freed larynx to one and to the other side, the trachea is now cleared of the tissues lateral to it by blunt dissection. Then a director is passed behind, between the trachea and the œsophagus, and here also some hæmorrhage may occur but can be ignored. The trachea is now to be cut across, and in order to secure it, four sutures are, first of all, passed through its walls, two above and two below the site of section; this done, it is cut completely across to the director behind it, the line of division passing upwards and backwards, so that the *upper* end of the thoracic trachea is left oblique. As the patient is breathing through the tracheotomy tube, the gaping upper end of the divided trachea (thoracic segment) is packed with a sponge for the time being, and the *lower* end of the laryngeal segment is held up by means of the sutures through it, so as to effect its separation from the œsophagus from below upwards. In this step the close fibrous adhesion between the posterior edge of the thyroid alæ and the prevertebral aponeurosis has to be cut through.

As this is being done, the superior laryngeal artery on the thyro-hyoid membrane is ligatured and divided, and then the upper attachments of the larynx to the pharynx and hyoid bone are cut through, and the entire thyroid and cricoid cartilages removed together with their contents. (Fig. 37.) The epiglottis is left.

The opening thus made in the pharynx is closely sutured, and the severed depressor muscles of the hyoid are brought together by catgut stitches.

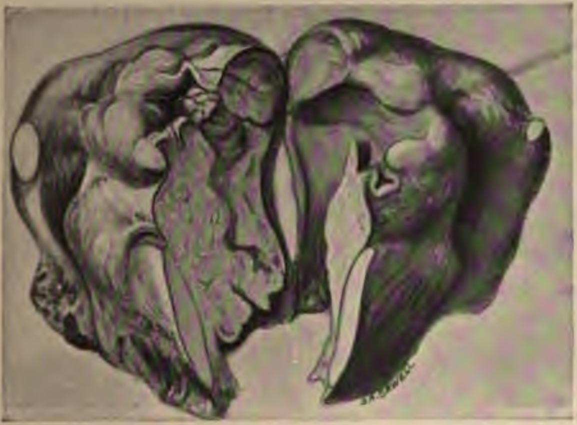

FIG. 37.—Larynx removed by Laryngectomy from Patient shown on Fig. 38.

The left half and part of the right half of the larynx is occupied by epitheliomatous growth.

The sponge in the upper end of the divided trachea is now removed, and the anterior lip of the trachea attached to the skin by sutures. Then its upper end is closed by iodized catgut sutures passed through it from side to side. The tracheotomy tube is retained in the former tracheotomy opening.

The skin is now sutured, the entire upper transverse incision being closed, and the median incision as far as the lower transverse wound; the lower transverse incision is partly united at the sides; the centre, containing the sutured and closed upper end of the trachea, being left open, is packed with iodoform gauze. A small feeding tube is passed through the nose into the œsophagus and left there.

After-treatment.—It is impossible to obtain a water-tight pharyngeal suture, and so, after the first day or two, mouth secretions find their way into the cervical wound, and discharges very freely; but as the lower part of the woun kept open, and the trachea is closed, all the discharges absorbed by the dressing in the lower wound.

In about a week, the discharge ceases, granulation is under weigh, and, the sutures in the upper end of the trac

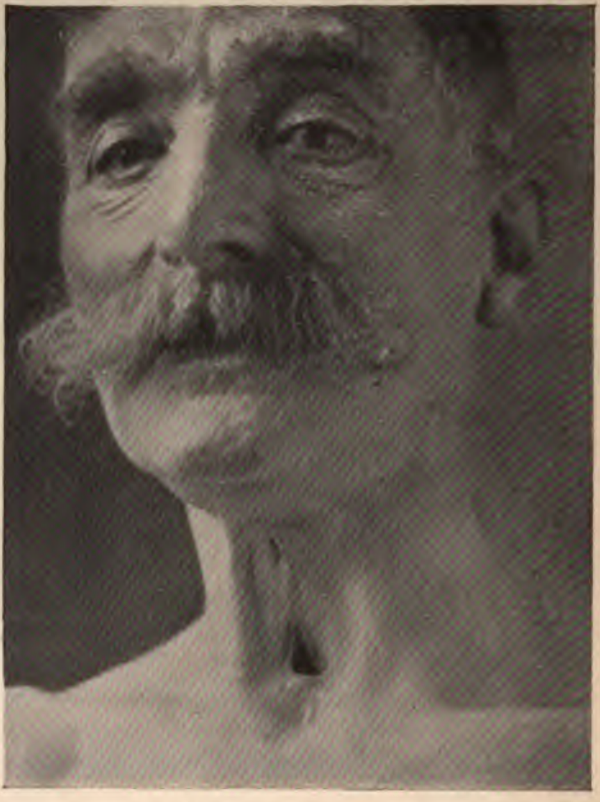

FIG. 38.—Patient after Laryngectomy.

See Fig. 37 for specimen removed. Untouched photograph taken five years a operation. No recurrence. The orifice of the trachea is clearly shown.

giving way, under the elastic tension of its cartilages, tracheal lumen opens up again.

A fortnight after the operation, the metal tracheotomy tu is removed from the tracheotomy opening, which is allowed close, and its place is taken by a long rubber tube inserted *i the upper end of the gaping trachea*, which, as the wound hea opens on the surface of the neck, being anchored in its new positi by scar-tissue. (See Fig. 38.)

The feeding tube is retained for ten days. All food is sterilized. The patient is encouraged to sit up in bed as soon after the operation as he can, and frequently washes out the mouth with an antiseptic mouth-wash.

Alternative Method.—The trachea is fully exposed at the preliminary operation, and cut completely across, the upper end of the thoracic or lower position being brought forward and stitched to the skin around its entire circumference. This step is performed a fortnight before the major operation. The drawback to this plan lies in the fact that the skin-tracheal sutures tend to give way before union is effected, and the patient, until the trachea becomes fixed, is rendered very uncomfortable by the loose tracheal stump, especially if there is much coughing.

The subsequent major operation is the same as the last, except that the trachea is not interfered with.

Results.—In cancer limited to the larynx, the chances of non-recurrence are good. But the operation, although nowadays fairly safe, exposes the patient to later risks of bronchitis and pneumonia. Nevertheless, many cases are now on record of prolonged life after laryngectomy. (See Fig. 38.) Those patients, although they are altogether voiceless, nevertheless often contrive to produce sounds in the pharynx, and to " speak," so as to be understood by those about them. Moreover, the use of an "artificial larynx" may still further aid the patient in this matter.

Sub-Hyoid Pharyngotomy.—The opening into the pharynx through the thyro-hyoid membrane by a transverse incision below the hyoid bone. (See Fig. 39.)

Indications.—Cancer of the upper orifice of the larynx, of the epiglottis, and in certain forms of cancer of the base of the tongue.

Operation.—Unless the larynx is obstructed by the growth, preliminary tracheotomy may be dispensed with, but the tracheotomy tube facilitates the administration of the anæsthetic.

The skin-inciison is transverse, passing at the level of the hyoid bone from the tip of the greater cornu on one side to the corresponding point on the other. The hyoid bone is exposed by deepening the incision and the depressor muscles are divided near their insertions so as to expose fully the thyro-hyoid membrane. All vessels having being seized, this membrane is now divided from one side to the other, close under the hyoid bone, so as to avoid injury to the superior laryngeal nerve. The underlying mucous membrane of the base of the tongue is now divided and bleeding vessels secured. This gives access to the pharynx in the glosso-epiglottic fossæ, the epiglottis and the rest of the larynx being in the lower part of the wound, and tumours here can be removed.

To close the wound, the mucous membrane is sutured first, then the muscles, and finally the skin. Provision should be made for drainage. The tracheotomy tube may be removed at once unless the operation has involved the larynx and there is any risk of reactionary hæmorrhage or of œdema of the glottis.

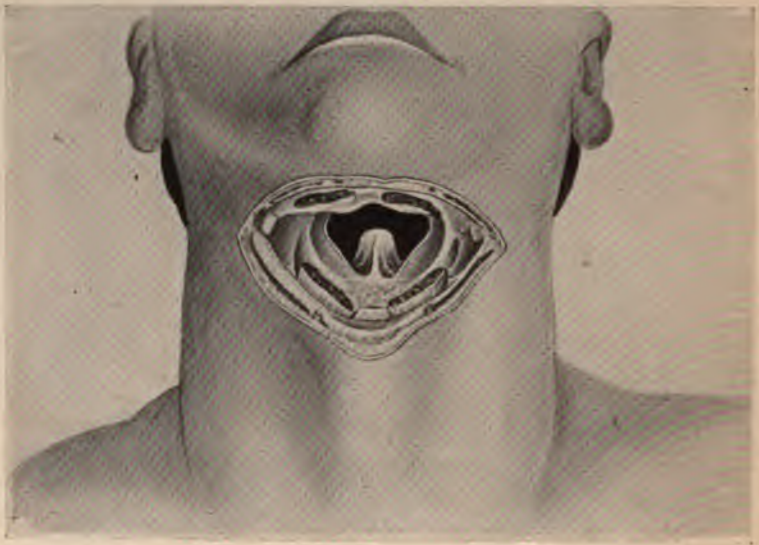

FIG. 39.—Sub-hyoid Pharyngotomy (after Kocher).

The after-treatment follows the same lines as that of laryngo-fissure. (See p. 140.)

TRANSTHYROID PHARYNGOTOMY

(Wilfred Trotter)

Description.—The opening of the pharynx through a lateral external incision and deep cervical dissection carried down to the hyoid bone and thyroid cartilage, the hyoid cornu and thyroid ala being excised to furnish access to the pharyngeal wall.

Indications.—Cancer in the epiglottic and arytenoid regions ("epilaryngeal" cancer), and post-cricoid ("hypopharyngeal") cancer. (See also p. 68.)

It is advised that when the disease is situated on one side of the pharynx, the exposure by operation should be made through the opposite side of the neck.

Preparation is the same as for laryngectomy. (See p. 141.)

Anæsthetic.—A preliminary tracheotomy is performed, and the anæsthetic is administered through the tracheotomy tube.

Operation.—The cervical incision runs along the line of the sterno-mastoid from just above the level of the angle of the jaw down to the level of the cricoid or further. The deep cervical fascia is divided from above downwards along a line an inch anterior to the skin incision, in order to provide a flap for subsequent use.

A second skin incision from about the middle of the first, and carried upward and forward towards the point of the chin, is also required.

The cervical dissection of the areas thus demarcated involves the removal of connective tissue, fat, and glands from the superior carotid and submaxillary regions, in the course of which the sterno-mastoid muscle is wholly or partially removed, and the external carotid artery ligated and divided.

Then the fascial flap described above is folded over the carotid vessels, which have been freely exposed by these manipulations, and stitched to the prevertebral fascia and muscles behind the pharynx. This is done in order to shut off the large blood-vessels from infection by the wound secretions of the subsequently opened pharynx.

The cricoid, thyroid, and hyoid are now cleared of their attached muscles—the inferior constrictor, stylo-pharyngeus, hyo-glossus, middle constrictor, etc. The pharyngeal wall is separated underneath from the deep surfaces of hyoid cornu and thyroid ala, and these structures, now completely isolated, are excised, the whole of the greater hyoid cornu and at least two-thirds of the thyroid ala being removed, together with the thyro-hyoid ligament connecting them. The intact pharyngeal wall now lies fully exposed.

Sutures are passed through this wall on either side of what is to be the incision through it, and when sufficient have been inserted, the wall is raised by their means, and a longitudinal incision made through it from above downwards. This manœuvre is necessary if we are to avoid soiling the neck wound with septic pharyngeal secretions.

The pharyngeal incision exposes the introitus laryngis; the base of the tongue and epiglottis; and below, the laryngo-pharynx.

The mouth and naso-pharynx having been plugged with sponges to hold up mucus from these cavities, the growth is examined, defined, and excised together with a free area of healthy tissue around it. The laryngeal wound thus made is closed with deep sutures, as is the pharyngeal incision.

Finally, the wound area in the neck is obliterated by suitable suturing, provision being made for free drainage, however, by means of large drainage tubes extending down as far as the pharyngeal incision.

After-Treatment.—Feeding is carried on through a nasal tube inserted at the operation, and the tracheotomy tube is left in position for a week.

The after-treatment generally is the same as that for laryngectomy. (See p. 146.)

For laryngotomy see p. 187.

NEUROSES OF THE LARYNX

Sensory.—Anæsthesia may arise from a *peripheral* nerve lesion such as neuritis from diphtheria, in which the superior laryngeal nerve or the vagus trunk, of which the superior laryngeal is a branch, is involved. It may also follow a traumatism of the superior laryngeal nerve, as in war-injuries, suicidal attempts, or operation.

In the neurotic type, motor laryngeal paralysis is common, and other cranial nerves may be affected.

Of the diseases of the central nervous system which produce laryngeal anæsthesia, the commonest are bulbar paralysis, locomotor ataxy, general paralysis of the insane, and hysteria. In these conditions the anæsthesia is almost invariably bilateral.

Symptoms.—In unilateral anæsthesia deglutition is often imperfectly performed, the food tending to enter the larynx, and to set up spasm and choking. Or, if the glottis is completely insensitive, as in bilateral anæsthesia, particles of food find their way into the trachea, and set up bronchitis and septic pneumonia.

Anæsthesia of the larynx is frequently combined with anæsthesia of the pharynx, and with motor paralysis of these parts.

The *prognosis* depends upon the cause. The diphtheritic type tends to recovery in about a month or six weeks, provided that the disease is not extensive. (See p. 38.) That due to injury of the superior laryngeal nerve may be permanent. And the bilateral type arising from a central nerve lesion is of serious import from its tendency to end in pneumonia.

Treatment.—Diphtheritic cases are kept in bed. Faradism is employed, and strychnine given hypodermically. (See p. 39.)

If the affection is bilateral and deglutition is impeded, the patient should be fed by a tube passed through the nose into the œsophagus. As the larynx is insensitive, make sure by the laryngoscopic examination that the tube is actually in the œsophagus and not in the larynx before passing any food through it!

Hyperæsthesia and Paræsthesia of the larynx are generally expressed by the patient as a feeling of pain, of tickling, of soreness, of rawness, or of the presence of a foreign body in the larynx.

Singers and voice-users are very prone to suffer from symptoms such as these, or from aching in the throat or neck, and from feelings of tiredness or weakness, especially after singing.

Diagnosis.—Careful examination is needed to eliminate a local cause, such as enlarged faucial or lingual tonsils, or early laryngeal tuberculosis. Sometimes a naso-pharyngeal abnormality will be found to be responsible for the disturbance.

If no local cause is present, search should be made for signs of tabes, seeing that attacks of pain or paræsthesia of the larynx with glottic spasm may really be the laryngeal crisis of tabes dorsalis. Sometimes the paræsthesia is hysterical, resembling the *globus hystericus.*

Treatment consists in removing any local abnormality, but such local treatment should only be adopted when there is some clear indication for it, as these patients, being often neurotic, are liable to become throat hypochondriacs.

If possible, firm reassurance that there is no serious local disease such as cancer or tuberculosis should always be given.

Periodical cocainizing and cauterizing the pharynx or dabbing the healthy larynx with local applications should not be encouraged.

Motor Neuroses of the Larynx. Hyperkinesis—Spasm of the Glottis.—Spasmodic attacks of choking or sensations of choking —*croup*—may arise from irritation of the laryngeal sensory nerves. (See table, p. 9.)

Spasm of the Glottis in Children may be due to a foreign body impacted in the larynx or loose in the trachea; to acute laryngitis, whether diphtheritic or simple (see p. 97); or it may arise from some reflex cause, in which case it is known as

Laryngismus Stridulus or **Spasmodic Croup.** This malady is commonly found in delicate or ill-nourished infants or young children, who are suffering from adenoids. Not infrequently, however, we are unable to determine the actual cause of the attacks, in which case it is inadvisable to take refuge in a diagnosis of " worms," or " teething."

Symptoms.—The attack comes on suddenly without premonitory symptoms, and usually during the night. It is frequently very alarming, inasmuch as it induces symptoms of asphyxia, which indeed in very delicate children have been known to end in death, but as a rule they subside spontaneously in a minute or two. After a period of crowing, cyanosis, and struggling, ending sometimes in a short spell of apnœa, normal easy breathing is restored, and the little patient falls asleep again, waking up in the morning either quite well, or with just a croupy cough as a reminder of the ordeal. But the attacks are very liable to recur, and may indeed attend every slight " cold " or catarrh.

Diagnosis.—Spasmodic croup is a-pyrexial, and it is further distinguished from inflammatory spasm or œdematous obstruction by its evanescent and recurrent characters. The first attack, however, may be difficult to diagnose correctly.

A series of recurrent attacks of glottic spasm within a brief space of time should raise the question of a loose foreign body in the trachea, and the patient should be X-rayed.

The child should also always be examined for adenoids, as laryngismus stridulus is most frequently merely an expression of these growths.

Treatment.—During the attack, the spasm may be at once allayed by applying a sponge wrung out of hot water—not too hot—to the front of the larynx. Rhythmical traction of the tongue is recommended. If the apnœic period were prolonged artificial respiration would be called for, and perhaps even intubation or tracheotomy might be necessary. But as a matter of fact, in genuine spasmodic croup in children the attack is at an end before even simple remedies can be applied, and if severer remedies prove to be necessary, the case really belongs to one of the more serious varieties of croup.

The best *preventive* of the attack is the removal of adenoids.

Spasm of the Glottis in Adults may occur without any definite cause, but in most cases a cause will be found if sought for. The spasm may be due to reflex irritation from the pharynx or larynx, as during laryngoscopic examination, or it may result from the inhalation of irritating or asphyxiating gases and vapours as in modern warfare. In these conditions it is, of course, non-recurrent.

As a recurring affection it may arise from irritating or acute maladies of the larynx; from the presence of a foreign body in the larynx; and it is occasionally induced by an elongated uvula or a hypertrophied lingual tonsil.

The early stages of recurrent laryngeal nerve paralyses are sometimes attended with glottic spasm, and so in the diagnosis of such cases the causes of recurrent laryngeal paralysis must be held in mind.

We have already mentioned tabes as a cause of glottic spasm in the laryngeal crisis, and it is occasionally hysterical in origin.

We may include here also the peculiar nervous condition known as *Ictus Laryngis* or *Laryngeal Vertigo*, which consists in a violent attack of coughing followed by glottic spasm and a momentary loss of consciousness; a group of symptoms obviously related to the epileptic *petit mal.*

The *Symptoms* require no special description. In the purely reflex and spasmodic cases the attacks are seldom dangerous to life though apt to be very alarming, especially if they make their first onset during the night.

The *Diagnosis* involves an exclusion of bilateral abductor (dilator) paralysis (see p. 155), and necessitates a consideration of the causes we have just synopized.

The *Prognosis* depends upon the cause.

Treatment.—Control of the larynx may sometimes be regained by deliberate, slow breathing, or simply by phonating. But in the severe and prolonged attacks, chloroform may be necessary to subdue the irritation, and the need for tracheotomy may arise.

STAMMERING AND STUTTERING.

though not strictly a laryngeal neurosis may be conveniently discussed here.

Stammering seems to be due to an imperfect co-ordination of the muscles which operate the mechanism of speech, respiratory, laryngeal and oral. The first supply the current of air which, acting along with the adductors and tensors of the larynx that bring the cords into apposition, produces the sound, while the modification of the sound by the muscles of the pharynx, soft palate, tongue and lips form vowel-sounds and consonants, syllables, words and sentences.

Any irregularity in the timing of these normally associated movements will result in hesitation, and so in stammering.

It must be added that this is not by any means the only theoretical explanation of the defect. It has been variously described as a lack of appreciation of consonants, especially initial consonants such as p or t, and on the other hand, as a lack of the kinæsthetic sensation of vowel-sounds. (" Kinæsthetic " ; i.e., sensation of movement.)

Whatever the analysis of the defect may ultimately prove it to be, the writer has no doubt that it represents an imperfect development of the faculty of speech, or, when it follows apoplexy or an aphasic attack, a stage in the disintegration of that faculty. The former is often hereditary.

Stammering is also common during recovery from the aphasia of shell-shock, and in this variety also it may become a permanent habit.

Varieties.—In addition to the common types of stammering over an initial consonant, we have the *silent stammer*, where the patient, although obviously labouring under stress, is aphasic for a few moments instead of stammering. Another more common variety is the interpolation of " ha " or " er " before the obstructing word or syllable.

Symptoms.—Stammering usually appears in childhood as the child is learning to talk, and once it is established, it tends to remain as a life-habit in spite of all efforts at cure. The stammer affects some words and syllables more than others, the most difficult being the explosive consonants, such as p and b, t and d. Stammerers, knowing their most difficult words in conversation, learn to employ periphrases in order to avoid encountering those obstacles.

The stammer is usually worse in company, especially in strange company, or when the patient is nervous or excited, and such patients are usually men (it is commoner in the male sex) of decidedly unstable nervous constitution. It is interesting that when the patient is alone, and when he is singing or reciting, the stammer is absent.

Prognosis.—Treatment may modify the habit, but it is seldom entirely curable.

Treatment.—As soon as a child is known to stammer, efforts should be made by parents and the teachers to instruct the child to practise slow speaking, and, especially, not to begin each uttered phrase or sentence until a comfortable, deep breath has been taken.

There are many " systems " in vogue for improving the established stammer, the favourite being to drop out the letter which is most troublesome ; thus, if " t " is difficult, the word " talk " is pronounced " alk " and so on. And a certain amount of improvement follows the use of such devices. The best results will be obtained when treatment is adopted early in life.

LARYNGEAL PARALYSIS

The whole of the intrinsic motor mechanism of the larynx is controlled through the inferior or recurrent laryngeal nerve, a branch of the vagus, with the single exception of the crico-thyroid muscle, which is supplied by the superior laryngeal nerve—the sensory nerve of the larynx.

Speaking generally, the lesion producing laryngeal paralysis may be situated—

1. In the cerebral cortex, or internal capsule. (Cortical laryngeal paralysis is nearly always hysterical, and it is always bilateral).
2. In the medulla oblongata, in the neighbourhood of the vago-accessory nuclei. Or it may arise
3. From intracranial pressure by tumour and syphilitic or other meningeal inflammation on the vagus trunk in its way to the jugular foramen, its route of exit from the cranium ; or from pressure on the vagus or its laryngeal branches in the neck or thorax, as by goitre, thoracic aneurysm, mediastinal new growths, etc.
4. From peripheral neuritis of the vagus or its branches.
5. From pressure on the nerve-endings in the muscles of the larynx in laryngeal inflammations and infiltrations—the myopathic paralysis.

The diagnosis of the different varieties of laryngeal paralysis can be made from the laryngoscopic appearances, but the diagnosis of their cause is a much less simple task.

Paralysis of the (Crico-Thyroid) Muscle supplied by the superior laryngeal nerve. This is a rare form of paralysis, and when it does occur it is generally associated with anæsthesia of the larynx.

(See p. 148.) The normal contraction of the crico-thyroid muscle draws down the thyroid cartilage over the cricoid, stretching the vocal cord and rendering it tense. When the muscle is paralysed the corresponding cord presents a wavy outline, and the voice is weakened, especially if the paralysis is bilateral.

The prognosis naturally depends upon the cause of the paralysis. When bilateral and combined with complete laryngeal anæsthesia, there is danger, the cough-reflex being abolished, of food passing into the bronchi and setting up septic pneumonia.

Paralysis of the Muscles supplied by the Recurrent Laryngeal Nerve (" Recurrent " paralysis).—When the recurrent laryngeal, the motor nerve of the larynx, undergoes gradual destruction in consequence of disease or pressure-atrophy of its fibres, the muscles under its control are paralysed, not haphazard or simultaneously, but in groups, according to their function. First, the abductors (or dilators) are affected ; next the internal tensors ; and lastly the adductors (or constrictors). As the abductors of the cord (or laryngeal dilators) suffer first, their action is abolished while yet the other groups are fulfilling their normal function. That being so, the adduction of the cords by their adductors and the resulting phonation is not interfered with in early recurrent paralysis, and thus when the paralysis is *unilateral*, the affected cord being retained by its active adductors in the middle line while the other cord can move normally, the patient will be able both to speak and to respire, and so no symptoms whatever will exist to direct his attention to the larynx.

Thus, the first stage of *unilateral recurrent paralysis* either escapes notice, or is discovered accidentally.

Bilateral recurrent paralysis, however, even in its earliest stages does attract attention, not, at this period, because of any interference with the voice, but because of the impediment to respiration produced by the vocal cords, which, by reason of the unopposed action of their adductors (constrictors of the larynx) come to lie near the middle line, and more or less in contact with each other. The glottis being thus closed, dyspnœa results, and asphyxia, and even death may occur as a consequence.

After the abductors (or dilators), the group of muscles next to be immobilized by the advancing destruction of the recurrent laryngeal nerve fibres is that of the internal tensors, Finally, the adductors of the cord (constrictors of the larynx) undergo paralysis, and with their involvement, recurrent laryngeal paralysis becomes complete.

(The above description applies to laryngeal paralysis due to *organic* lesions of the motor nerves. On the other hand, it is found that if the adductors (constrictors) or tensors are affected, while the abductors (or dilators) are moving normally, then the paresis of paralysis is *functional* (hysterical) or *myopathic*, and not due to an organic lesion of the nerve.) (See later, p. 160.)

We proceed now to discuss paralysis of the several muscle groups in detail, and then we shall go on to describe the laryngeal appearances in total recurrent laryngeal paralysis.

The first stage of recurrent paralysis is **Abductor (Dilator) Paralysis.**—(The abductor of the vocal cord is the crico-arytenoideus posticus.)* This is commonly produced by pressure on the recurrent laryngeal nerve or nerves by tumours, aneurysm, goitre, enlarged mediastinal glands, or by malignant disease of the œsophagus. A central nerve lesion in the medulla or of the vagus at the base of the brain, such as may occur in tabes, bulbar paralysis, disseminated sclerosis, or intracranial syphilitic disease, may be responsible for the paralysis, or it may arise from herpes or from neuritis of toxic origin.

Among the rarer causes we may mention enlarged tuberculous intrathoracic glands; pleuritic adhesions about the apex of the right lung, implicating the right recurrent laryngeal nerve; and dilatation of the heart.

In a certain proportion of the cases seen, no cause can be found for the paralysis.

As a rule, abductor paralysis is unilateral, and is due to some lesions exercising pressure on the trunk of the recurrent laryngeal in its course from the thorax to its termination in the larynx.

As we have already seen, *in unilateral abductor paralysis* the cord takes up a position in the middle line from the unopposed action of the adductors. Thus phonation is not affected, and the discovery of the phenomenon is, as a rule, accidental.

When the *abductors of both cords are paralysed*, the cause is to be found either in large thoracic tumours commanding both recurrent laryngeal nerves, in some central lesion, or in some condition, toxic or otherwise, which tends to produce symmetrical disturbances.

In bilateral abductor (dilator) paralysis, as we have said, both of the cords occupy a median position, lying more or less in contact. There is no hoarseness, but there is dyspnœa, and, frequently, loud stridulous breathing (laryngeal stridor). As the tensors and finally the adductors (constrictors) become paralysed the dyspnœa and stridor tend in due course to disappear, but, to begin with, the interference with respiration may be so great as to necessitate tracheotomy, and the wearing of a tracheotomy tube for a time.

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* Hence the name sometimes given of "posticus" paralysis:—The atrophy of the paralysed "posticus" muscle is well exhibited in the Frontispiece.

It is characteristic of these cases that if the paralysis is of very gradual onset the patient becomes so accustomed to the impediment that he himself may be able to ignore, or indeed may be actually unaware of any dyspnœa or difficulty in breathing. And an amount of obstruction which if suddenly produced would cause asphyxia and perhaps death, is tolerated with scarcely any resentment. The only difficulty experienced is on exertion, or during an attack of laryngitis or bronchitis.

Nevertheless, the danger, though ill-appreciated, is real enough, for a slight increase in the obstruction, as from an acute laryngitis, is sufficient to tilt the balance and to lead to rapid death.

For that reason, immediate tracheotomy should be urged upon the patient, but, as a matter of fact, unless he has had actual experience of a real dyspnœic attack, he will be hard to convince of the necessity for the operation.

Paralysis of the Internal Tensors.—(The internal tensors of the vocal cord are the thyro-arytenoideus internus and externus, which are supplied by the recurrent laryngeal nerve. The external tensors are the crico-thyroidei supplied by the superior laryngeal nerve; see *ante*, p. 153.)

When internal tensor paralysis occurs in association with abductor (dilator) paralysis *in the second stage of recurrent paralysis*, the cord still occupies the median position, but the voice is weak as the approximation of the cords is imperfect.

Complete paralysis limited to internal tensors alone, and due to a nerve lesion is very rare. When it does occur, it is said that abduction (dilation) occurs as in health; but on attempting phonation, the cords only come together along their posterior third (from the unaided action of the adductors), and fail to meet in their anterior two-thirds.

Bilateral paresis or paralysis of the internal tensors is most commonly functional, due either to hysteria or to myopathy, as in chronic laryngitis.

Adductor (Constrictor) Paralysis.—(The adductors of the vocal cords are the crico-arytenoidei laterales which, by rotating the arytenoid cartilages, bring their inner angles, bearing the cords, together. In this they are aided by the arytenoideus muscle which approximates the arytenoid cartilages as a whole, and also by the thyro-arytenoidei externi.)

When in the advancing destruction of the fibres of the recurrent laryngeal nerve, the complete interruption of its conductivity is reached, paralysis of the adductors of the cord is added to the existing paralyses of the abductors and the internal tensors, and we have now to deal with *complete recurrent paralysis*, or as it is generally termed ," paralysis of the vocal cord."

This gradual extension of paralysis from one group of muscles to another is only seen when the conductivity of the nerve is being slowly and progressively interrupted, as when it is stretched and atrophied by an enlarging thoracic aneurysm or mediastinal tumour. When the nerve is suddenly and completely destroyed, as by a bullet wound, or by a surgeon's knife, in a goitre operation for example, total recurrent paralysis is produced instantaneously.

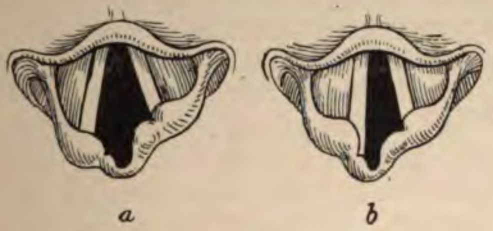


FIG. 40.—Recurrent Laryngeal Paralysis (Fixation of the Left Vocal Cord)—(*a*) during quiet respiration; (*b*) during attempted phonation. The usual over-adduction of the sound cord on phonation is not shown in this diagram.

In **total recurrent paralysis of one cord**, the cord assumes the cadaveric position, and is quite immobile. During phonation, the sound cord of the other side crosses the middle line in an effort to come into contact with the paralysed one, the arytenoid eminence of which may be seen during this movement to be pushed aside and behind by the healthy over-adducted arytenoid. In this way a certain amount of vocal tone is produced, and as time passes, the voice may gain in power and volume to such an extent that the patient may actually be able to speak in public. During quiet inspiration, the larynx appears normal. During deep inspiration, the sound cord alone is fully abducted, while the paralysed cord remains motionless in the position of partial abduction. (See Fig. 40 and Plate I.—Frontispiece.)

It is sometimes difficult to determine whether a cord moves or not, and thus paralysis is not infrequently overlooked.

To sum up what we have been saying :

In the first stage of recurrent paralysis, the abductors (dilators) alone are paralysed, and the adductors (constrictors) being unopposed, the cord is fixed in the middle line. If unilateral, the voice is unaffected. But if the paralysis is bilateral, there is stridor and dyspnœa.

In the second stage, paralysis of the internal tensors is added to paralysis of the abductors. The cord lies close to the middle line, but its approximation to the other in phonation is imperfect. The voice is weak.

In the third and final stage, paralysis of the adductors (constrictors) is added to paralysis of the abductors (dilators) and of the internal tensors. The cord lies motionless in the cadaveric position. There is hoarseness and imperfect cough.

The *course of the recurrent laryngeal nerve* must be considered in connexion with laryngeal paralysis, as the great liability of that nerve to pressure from tumours, etc., renders such a lesion the most common cause of these paralyses.

The nerve runs a somewhat different course on the right from what it does on the left side. Both are branches of the vagus. On the right side the nerve arises in the lower part of the neck. Coming off from the vagus as that nerve passes down in front of the first part of the subclavian artery, the right recurrent laryngeal loops below and ascends behind the subclavian artery ; it then passes upwards and inwards behind the common carotid and inferior thyroid arteries to gain the angle between the œsophagus and trachea, where it continues its ascent until it enters the larynx beneath the edge of the inferior constrictor muscle. The nerve of the left side arises in the thorax from the vagus as it lies in front of the arch of the aorta. It arches round below and then up behind the aorta, ascending into the neck behind the left subclavian artery. Having gained a position between the œsophagus and trachea, it passes up to enter the larynx under the lower edge of the inferior constrictor muscle. It does not run so close to the œsophagus as its neighbour of the right side. (See Frontispiece.)

As a result of this difference in position, the left recurrent laryngeal is more liable to pressure in aortic aneurysm and thoracic tumours generally, while the right recurrent is more frequently involved than the left in malignant disease of the œsophagus.

Diagnosis.—The differential diagnosis between a vocal cord fixed by ankylosis of the crico-arytenoid articulation and a paralysed cord has already been discussed. (p. 112.)

In the diagnosis of the cause of paralysis of the vocal cord in any particular case it is necessary to examine the neck for goitres or other tumours, and the chest for aneurysm or mediastinal tumour, in a search for which X-ray examination is indispensable.

As a result of the recurrent paralysis, generally unilateral and left-sided, the laryngeal symptoms of *aneurysm of the thoracic aorta* are: (1) cough, (2) paroxysmal dyspnœa, and (3) alteration in the voice. The cough is the well-known "brassy," "bovine," or incomplete cough produced by the partial closure of the glottis in coughing efforts; the paroxysmal dyspnœa is due to attacks of glottic spasm; and the hoarse voice to the immobility of the cord. "Tracheal tugging" may also be felt. It is elicited by getting the patient to extend the head fully so as to render the trachea prominent in the neck, and to put it on the stretch. In this position, communicated pulsation from an aortic aneurysm is readily detected by placing the thumb and one finger on the pomum Adami, and pressing it slightly upwards.

Bilateral Recurrent Paralysis (total laryngoplegia) is extremely rare. In this type, both cords are affected; they lie fixed in the cadaveric position, and do not meet under any circumstances. The patient is voiceless, and even the whisper can no longer be produced.

It is a curious fact that during deep inspiration, the paralysed cords may be seen to approach each other.

Prognosis of Recurrent Paralysis.—The outlook naturally depends upon the cause of the paralysis. Those forms which are due to neuritis often end in recovery, but those due to pressure on the nerve-trunk are usually permanent.

The prognosis, as regards vocal power in unilateral paralysis, is, as we have already seen, more favourable than might be expected. But as a rule, the patient remains permanently aware of some weakness in the voice.

Treatment.—The treatment must be applied to the cause. If that can be successfully overcome, the recurrent paralysis will disappear unless the damage to the nerve trunk is irreparable.

Paralysis of the Arytenoideus Muscle alone is not uncommon. It is usually either hysterical or myopathic.

On phonation the cords come together along their anterior two-thirds, but a triangular chink is left between the vocal processes (contrast with paralysis of the internal tensors).

The *Treatment* is applied to the cause.

Bilateral Adductor Paralysis (Functional Aphonia; Hysterical Aphonia).—Bilateral paralysis of the adductors of the cord, the abductors being active, is never due to recurrent paralysis. It is either myopathic from laryngitis, or hysterical in origin.

The *myopathic type* is easily diagnosed on examination. The cords being red and thickened, when efforts at phonation are made, they come into contact in front and behind, but leave an elliptical opening between them through which the air rushes, producing a sound that is little more than a whisper.

It is, perhaps, scarcely accurate to speak of this condition as a paralysis or even as a paresis since it is obviously due to the infiltration mechanically impeding the contraction of the muscular fibres of the cord.

The *Treatment* is that of chronic laryngitis. (See p. 108.)

Functional or Hysterical Aphonia is most commonly found in young women, but the Great War has made us acquainted with the same condition in an extremely obstinate form in men who have been exposed to shell-explosion or to other forms of severe shock.

The only symptom, apart from the stigmata of hysteria, is voicelessness, usually of quite sudden onset. In civil life it is predisposed to by local conditions, such as chronic laryngitis, and by constitutional asthenia as from anæmia, incipient phthisis, neurasthenia, or boredom. Its occurrence is frequently determined by some kind of shock or fright, and a shock or fright may also lead to its cure.

It is possible often before examining the patient's larynx, to hear a certain amount of phonation when the patient is made to cough. But in the most extreme cases the patient is also unable to cough, and even a whisper is beyond his powers.—(Apsitheiria.)

On laryngoscopic examination, the larynx, unless it is the seat of laryngitis, appears to be quite healthy in colour and form. The vocal cords move with respiration, but on asking the patient to phonate, the observer will mark that although they approximate, they fail to close, and only a noisy breath-sound is produced. At this moment, if the patient is made to give a gentle cough, the cords will be seen to come together to do so, thus clinching the diagnosis.

As we have already indicated, however, in shell-shock cases even the effort at coughing may be feeble and imperfect.

Diagnosis.—The possibility of early phthisis must not be forgotten.

Prognosis.—The voice will be restored again, either by treatment or spontaneously. But the aphonia may last for months and even for years. It is, moreover, a hysterical phenomenon, and on its disappearance some other hysterical paralysis may take its place. Recurrence, also, is common.

Treatment.—In first attacks one is often able to bring the voice back by moral suasion and the suggestive influences of the laryngeal mirror and examination. If we fail to do so at the first attempt, some other method should be tried, without any loss of hopeful confidence being made manifest.

The faradic current, fairly strong, applied to the neck or with one electrode in the larynx, is a very effective shock treatment. But it is painful and frightening, and is moreover open to the drawback that if it fails the malady seems to become more firmly rooted than ever. It is contra-indicated in shell-shock, sufferers from which are almost invariably made worse on exposure to violent and sudden stimuli.

For this class of patient, the administration of ether, as if for operation, combined with suggestion before and during the induction period, as well as when the patient is emerging from the anæsthetic, is frequently successful in restoring the voice.

Hypnotic suggestion also is sometimes of benefit (Burnett Rae), and especially the suggestion, apart from hypnotism, that emanates from a confident and persuasive personality (H. Smurthwaite).

There are, nevertheless, some cases which resist all methods, or where the success obtained is merely temporary. For such cases, tonics and good food to increase the bodily vigour should be given, and then the moral methods may be tried again with more chance of success.

The following table indicates roughly the causes of **Fixation of the Vocal Cord :**

1. *Disease or Ankylosis of Crico-arytenoid Articulation :*
    - (*a*) Rheumatic.
    - (*b*) Syphilitic.
    - (*c*) Tubercular.
    - (*d*) Carcinomatous.

2. *Perichondritis:*
    - (*a*) Traumatic.
    - (*b*) Syphilitic.
    - (*c*) Tubercular.
    - (*d*) Carcinomatous.
3. *Cicatricial Adhesions:*
    - (*a*) Septic.
    - (*b*) Syphilitic.
    - (*c*) Traumatic.
4. *Paralysis of Cord.*
5. *Contusion or fracture of thyroid cartilage.*

LARYNGEAL STENOSIS

The following table is a synopsis of the diseases of the larynx which cause narrowing of the lumen and obstruction to the air-passage.

The reader is referred for details to the section dealing with each item :—

I *Intralaryngeal Causes:*
  1. Swelling of soft parts from
      - (*a*) Acute laryngitis or perichondritis.
      - (*b*) Œdema, acute or chronic.
      - (*c*) Tubercular, lupoid, or gummatous deposits.
      - (*d*) Neoplasms, simple or malignant.
  2. False membranes, septic or diphtheritic.
  3. Laryngismus stridulus, and spasm of the glottis.
  4. Congenital laryngeal stridor
  5. Congenital web of the larynx, cicatricial adhesions and bands from traumatism ; gun-shot wounds ; high tracheotomy ; intubation.
  6. Ankylosis of the crico-arytenoid articulation.
  7. Bilateral abductor (dilator) paralysis of the cords.
  8. Foreign bodies in the larynx.

II. *Extralaryngeal Causes* (rare) :

Large foreign bodies in the œsophagus.

Symptoms.—In *laryngeal stenosis* there is stridor attending inspiration chiefly or exclusively. In *tracheal stenosis* the stridor is both inspiratory and expiratory. The loudness and quality of the adventitious sound vary with the nature and the degree of obstruction, and the same factors influence the symptom of dyspnœa. In this matter, also, the nature of the onset is of importance. Obstruction arising suddenly is badly borne, and tends to give rise to alarming symptoms, whereas the same amount of obstruction of gradual onset is tolerated without difficulty; and dyspnœa may only be noticed when the patient is making exertion, or during sleep.

The attitude instinctively adopted by the patient as being the least oppressive in laryngeal stenosis differs from that adopted in tracheal stenosis.

In laryngeal cases the head is thrown back; in tracheal cases it is held forward with the chin depressed so as to relax the trachea. The voice, in laryngeal cases, save in bilateral abductor paralysis, is rough and hoarse. In uncomplicated tracheal stenosis on the other hand, the voice is clear though weak, or it may, as in goitre, assume a " reedy " or bleating quality.

The prognosis and treatment of laryngeal stenosis depends upon the cause, and may be sought for in the sections dealing therewith. At present, we shall allude only to the treatment of cicatricial stenosis.

Cicatricial laryngeal stenosis is the result of old syphilitic disease, of gunshot injuries, or of high tracheotomy. A congenital web of the larynx extending between the cords and occupying the interval of the anterior commissure is occasionally met with.

We have already considered the subject of syphilitic scarring and contraction. (p. 113.)

Bullets passing through the larynx from side to side induce a wound which, if the patient survive, is apt to cause serious and permanent cicatricial stenosis, necessitating the continuous wearing of a tracheotomy tube. On examination, the upper surface of the mass of scar-tissue may look as if it were only a thin web, but the appearance is deceitful; and any attempt to open up the obstructed lumen by a simple intralaryngeal incision will fail.

The commonest type of cicatricial stenosis in civil life is that due to an unduly high tracheotomy, albeit it is less common than it used to be when tracheotomy was more frequently performed than it is to-day.

In the haste and excitement of urgent tracheotomy on infant or young child, the operator, particularly if he is worki with a poor light and without skilled assistance, has seldom ti or inclination to trouble about such refinements as the exa level of the opening he is making into the trachea. Consequentl it is occasionally found after an operation otherwise quite succes ful that although the disease is completely recovered from, i is impossible to remove the tube, because the child become asphyxiated each time the attempt is made to do so, through his being still unable to breathe through the larynx.

The cause of this inability lies in the fact that the tracheotomy opening is situated too near to the larynx, and the irritation of the tube on the thick submucous tissue of the larynx sets up an inflammatory œdema, with, later, granulation tissue formation which finally becomes scar-tissue, occupying the greater part of the lumen of the air-way. Moreover, if the patient is a child, and nothing is done to remedy the trouble, the larynx will fail to develop, and will remain undersized and infantile in later life.

If at the tracheotomy care be taken not to carry the incision in the trachea any higher than the lower border of the cricoid cartilage, this *contretemps* will never arise. It is, as a matter of fact, generally due to an omission to extend the head sufficiently while performing the operation. (See Tracheotomy, p. 178.)

Treatment.—The performance of a low tracheotomy is the first step, and in recent cases the only step necessary. No further treatment is required, as the laryngeal obstruction will disappear spontaneously.

In old cases, after a low tracheotomy has been performed, the larynx may be dilated by means of Schrötter's bougies, or by the wearing for prolonged periods of graduated vulcanite intubation tubes. If these plans fail, laryngostomy—the re-formation of the laryngeal lumen—may be carried out. But this operation and its after treatment are extremely tedious and the results do not seem to be particularly good.

For traumatic cicatricial stenosis of the larynx it suffices generally to divide the scar-tissue intra-laryngeally and to maintain the passage thus made by periodical dilatation.

The chances are favourable unless portions of the cartilaginous framework of the larynx have been lost.

Foreign Bodies in the Larynx, if large enough to block the airway, will naturally cause immediate death from asphyxia if not promptly removed. The symptoms are those of asphyxia with violent efforts at respiration. At such a moment, treatment must be speedy. The finger should be passed into the larynx, and the foreign body hooked out.

If attempts at removal fail, laryngotomy or tracheotomy should at once be performed, with a pen-knife if there is nothing better to hand. (See p. 182.)

Bodies too small to cause asphyxia may, if sharp or pointed, be arrested in the larynx, in which case they cause spasm, stridor and aphonia. The spasm may be violent enough to cause death, but impaction between the cords may actually prevent their spasmodic closure, and so maintain the airway. After a time the spasm subsides, and the larynx comes to tolerate the presence of the foreign body, so that it has been known to remain *in situ* for months, as in a case in a young child recorded by the writer. (Fig. 41.)

FIG. 41.—Rabbit-bone (actual size) removed from the larynx of a child two years of age by means of suspension laryngoscopy. It had lain impacted in the larynx for three months.

Treatment.—Tracheotomy first of all having been performed, the foreign body is removed in children by suspension laryngoscopy; in adults by direct laryngoscopy

CHAPTER VI

THE EXAMINATION AND AFFECTIONS OF THE TRACHEA, THYROID BODY, AND ŒSOPHAGUS

THE TRACHEA

The Examination of the Trachea.—The course of the trachea in the neck can usually be made out by external examination. Even in cases of difficulty where the neck is short and fat, for example, as in children, palpation can almost always be relied upon for correct information. The most difficult cases are those in which the trachea has been buried beneath a large goitre involving the isthmus of the thyroid, or beneath the diffuse brawny swelling of extensive cervical cellulitis.

The laryngoscope frequently affords us information concerning the shape and disposition of the trachea, which in favourable cases may be visible in the mirror as far as the division into the right and left bronchus. By making the patient stand up while the surgeon sits and practises laryngoscopy, it is possible to inspect the trachea as far as the carina.

Tracheoscopy and Bronchoscopy.—The tubes used for the direct examination of the trachea and bronchi are telescopic, and they are longer and finer than the laryngeal tube spatula. (See Fig. 42.) In length they vary from 35 cm. to 15 cm.; and in diameter from 14 mm. to 4 mm., to suit different ages.

In adults, local anæsthesia (see p. 92) is sufficient, but in children most operators will find a general anæsthetic to be necessary, although Chevalier Jackson is able successfully to dispense with it.

Position.—The patient, if an adult, is seated in the ordinary examining chair; on a low stool; or (always in children under an anæsthetic, and sometimes in adults) is laid flat on a table, with the head level and turned a little towards the examiner, or with the head extended over the end, in which case the surgeon seats himself at the head of the table.

A useful position is Mouret's, with the patient sitting stride-legs over a chair facing the back of it, the pelvis being pushed well back, and the chest resting against the back of the chair.

Method.—If there is severe dyspnœa, a tracheotomy should be performed before attempting to pass the bronchoscope. In that case it may be inserted through the tracheotomy opening (lower bronchoscopy). Otherwise, the tube is passed through the mouth and larynx. (Fig. 42.)

The first steps are the same as for direct laryngoscopy. (See p. 87.) After the tube has passed the epiglottis and is turned down into the larynx, at which moment the proximal end will occupy a position at one or other angle of the mouth, by gentle pressure, and under inspection the distal end of the tube is borne through the glottis between the vocal cords, and so onward down the trachea to the bifurcation, which is easily recognized. To enter either main bronchus, the tube is diverted slightly to the one or other side, the handle meantime moving to the side of the patient's mouth opposite to the bronchus which we desire to enter.

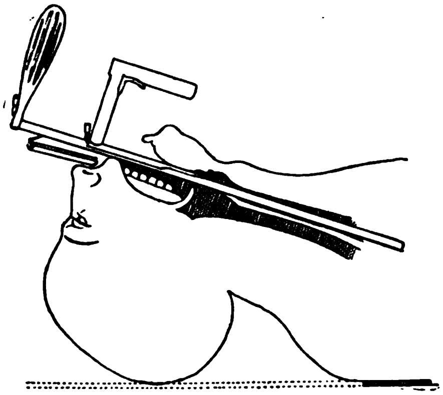


FIG. 42.—Bronchoscopy. The passage of the bronchoscope through the direct laryngeal tube—(Chevalier Jackson).

The bronchi may be painted with 10 per cent. cocaine solution.

Passing on in this way, we inspect the lumen and walls of the trachea and main bronchi and are able to penetrate as far as to the bronchi of the second and third degree.

Difficulties and Dangers.—The passage of the bronchoscope on an adult by skilled hands, working with skilled assistants, is not difficult or dangerous in itself. The difficulty and dangers lie in the circumstances which call for the operation, such as the presence in trachea or bronchus of a foreign body.

In infancy and childhood, however, upper bronchoscopy must be rapidly, though not forcibly performed, as the presence of the tube in the larynx for longer than fifteen minutes will be followed by laryngeal œdema necessitating tracheotomy, and for the same reason, on infants and children one uses the finest possible tubes.

Bronchoscopy is performed for the removal of foreign bodies (see p. 189), and, more rarely, for the removal of tracheal neoplasms. Asthma has been treated by direct application of remedies to the bronchi through the bronchoscope.

DISEASES OF THE TRACHEA

Displacements.—The trachea may be deviated to one or other side by tumours such as unilateral enlargement of the thyroid, or by inflammatory swelling in the neck. Such deviations can quite frequently be made out on examination with the laryngeal mirror.

Tracheal Stenosis is caused by:

I. *Intratracheal Causes:*

1. Gummatous infiltration.
2. Syphilitic cicatrices.
3. Swelling of the mucosa from irritating vapours, etc.
4. Stricture following tracheotomy.
5. Neoplasms.
6. Foreign bodies in the trachea.

II. *Extratracheal Causes:*

1. Enlargement and tumours of thyroid gland.
2. Traumatism (wound, fractures).
3. Cervical cellulitis.
4. Aneurysm (aortic or innominate).
5. Enlargement and tumours of the thymus.
6. Growths and foreign bodies in the œsophagus.
7. Tuberculous bone abscess from vertebræ, sternum, etc.

Gummatous infiltration of the trachea leads to constriction and is dangerous to life if its formation is not checked or if it is situated in the thorax.

The nature of the infiltration may be surmised by the presence of syphilitic signs elsewhere, or by the Wassermann reaction being positive.

Treatment by one of the salvarsan group should be at once undertaken, and mercury given intra-muscularly. The patient should be kept in bed. Healing is sometimes followed by cicatricial stenosis.

Neoplasms in the trachea are rare. But several have recently been recorded, some of them simple (papilloma—W. H. Kelson), but the majority epitheliomatous or endotheliomatous (Seymour Jones).

There is a tendency for malignant growths in tissues adjoining the trachea, such as the thyroid, to penetrate the tracheal wall, and to give a false appearance of genuine intra-tracheal neoplasm, and error is facilitated by the fact that the intra-tracheal extension is frequently the first indication given of the extra-tracheal neoplasm. The microscopic appearances of the intra-tracheal tumour after its removal usually inform us, however, of its true nature and origin.

Symptoms.—Progressive difficulty in breathing is the first complaint, and the dyspnœa, which is aggravated by exertion, is accompanied by tracheal stridor with recession of the episternal notch, and, if severe, also of the intercostal spaces. Persistent cough with paroxysmal exacerbations is common, in the course of which pieces of the tumour may be expectorated.

In uncomplicated cases of genuine intra-tracheal neoplasm, the trachea externally shows no deviation or distortion, but on direct examination, the growth is at once obvious. A piece should be removed for microscopic examination in order to exclude extra-tracheal tumour.

Prognosis.—If entirely removed, simple growths do not recur, but the prognosis in malignant tracheal growths is unfavourable.

Treatment.—If situated above the level of low tracheotomy, that operation should be performed and a tracheotomy tube inserted. Thereafter the trachea above may be exposed, incised, and held open by retractors while the neoplasm is eradicated.

Tumours below this level are removed as thoroughly as possible through the direct tube.

(For Foreign Bodies in the trachea, see p. 189.)

THE THYROID GLAND

Only those diseases of the thyroid gland will be touched upon which have particular bearing upon laryngology.

The normal thyroid gland is not very obvious on palpation, only an indefinite fullness or thickening being detectable in front of the trachea, where the isthmus occupies a position from the second to the fourth tracheal ring—i.e., about half an inch. The lobes also are inappreciable in health, being overlaid by the sterno-mastoid, sterno-hyoid, and sterno-thyroid muscles.

Attached to the upper margin of the isthmus, the pyramidal lobe, developed from the thyro-glossal tract, may sometimes be felt.

The thyroid gland being adherent to the trachea and larynx, moves with them in deglutition, an important point in the distinguishing of goitre from other cervical tumours

AFFECTIONS OF THE THYROID

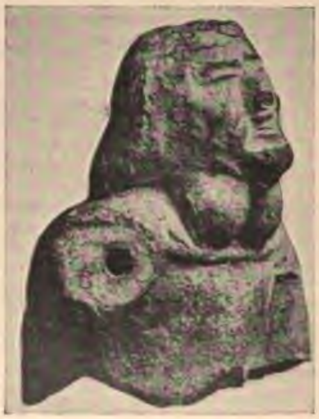


FIG. 43.—Votive offering showing Goitrous Enlargement of the Neck, found on the Island of San Bartolomeo (Rome) and referred to Etruscan times, about 2000 B.C. (From Messrs. Oppenheimer & Co.).

Any enlargement or tumour of the thyroid body may be termed "goitre"; there are three varieties of goitre: simple, exophthalmic, and malignant.

Simple Goitre is a general hypertrophy of the normal parenchyma of the gland, which, in its endemic form, the writer showed many years ago to be probably brought about by an effort to counteract a toxin produced by an organism introduced into the body by means of infected drinking-water.

Recent investigations and experiments by McCarrison have placed this theory beyond doubt.

Simple parenchymatous enlargement may produce a goitre of very considerable size. In its early stages it reveals itself in a fullness in the lower neck of the general shape of the thyroid gland. It is most commonly found in women, and generally first appears about puberty. The right lobe is usually larger than the left.

A thyroid which has undergone simple hypertrophy is very prone to secondary degenerative changes, which may result in the formation of a huge tumour seriously affecting the neighbouring organs and structures by pressure.

These secondary changes are as follows:

1. Fibroid degeneration, forming *Fibrous Goitre*, which may be associated with myxœdema from sclerosis and atrophy of the secreting acini;
2. *Colloid Goitre* where the follicles are dilated and much colloid material is present.
3. One or more of these dilated follicles may enormously enlarge to produce *Cystic Goitre*.
4. One or more areas in the gland may become the seat of adenomatous growths, the adenomatous tissue being largely composed of embryonic thyroid tissue —*Adenoma of the Thyroid*.

In endemic areas *congenital goitre*, and *acute goitre*, the latter of which is marked by rapid enlargement of the whole gland in a few days or weeks, may also be met with.

General Signs and Symptoms.—As a rule, the gland enlarges very gradually, and the enlargement affects lobes and isthmus alike. The early parenchymatous goitre feels soft on palpation, but in the course of months or years, the secondary changes which occur lead to an alteration in the physical characters of the tumour; thus in the larger parenchymatous and other varieties the gland as a whole is firm from the fibrous changes which are present in nearly all cases.

The pressure of the enlarged gland leads to an atrophy of the overlying muscles, to a backward displacement of the carotid sheath with its vessels and nerves, and to a marked and tortuous stenosis or flattening of the trachea. Pressure on the œsophagus may lead to dysphagia, and compression of one or both recurrent laryngeal nerves to laryngeal paralysis.

In the cystic form, large tense tumours may be felt, differentiated from the main mass of the goitre by their smooth, hard walls.

In adenoma occurring in a goitre, or in an otherwise normal thyroid, the tumour is nodular, and rounded.

The most serious symptoms arise from displacement and stenosis of the trachea. Stridor is marked and dyspnœa usual.

Diagnosis.—Simple goitre must be distinguished from exophthalmic goitre and from malignant goitre. A glance at the account given of these two diseases will enable the student to make a correct diagnosis in most cases.

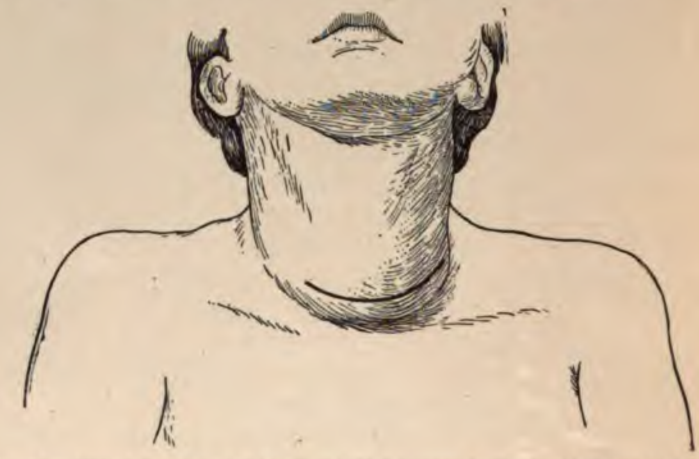


FIG. 44.—Collar Incision for Removal of Adenoma or Lobe of Thyroid Gland (after Kocher).

The *prognosis* depends upon the age, the rate of growth, and the size of the enlarged thyroid. Recent slight enlargements may be kept within bounds by simple treatment, and goitres of long standing, which cause no pressure symptoms, and are not increasing in size, may be ignored.

Treatment.—Early goitres may be successfully treated by thyroid tabloids internally, provided there is no tachycardia, from 3 to 5 grains of the gland substance being given at bedtime. Locally, treatment by the iodine compress may be tried. It is a piece of lint, three inches by two, soaked in

℞.	Tc. Iodi.,	ʒ i.
	Glycerin.	ʒ ii.
	Aq. ad.	℥ i.

applied over the goitrous swelling and covered with oilskin held in position by a bandage round the neck. This application is made every night until the integument begins to peel, when it is intermitted until the skin is well again, and then repeated. In endemic areas the drinking water should be boiled and personal cleanliness insisted upon.

Surgical removal of a portion of the gland is called for if the goitre is causing dyspnœa or stridor, or if it is rapidly enlarging; the clinical rule being to operate if the patient is unable to breathe comfortably when lying flat. Adenomata are often removed simply to remedy deformity.

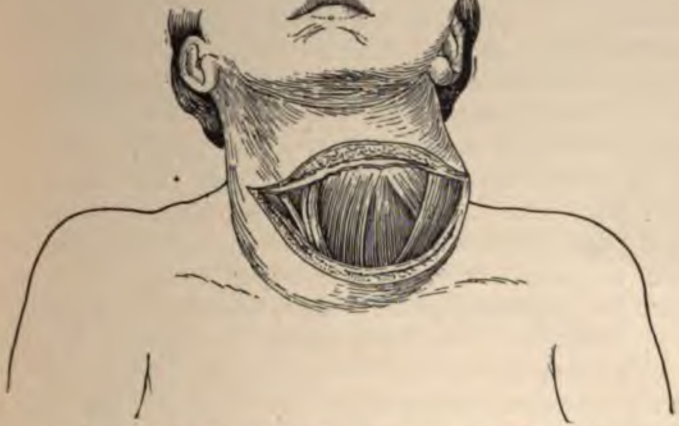


FIG. 45.—Thyroidectomy. Exposure of muscles through the collar incision (after Kocher).

Operation (1) *For Adenoma.*—A curved "collar" incision from two to three inches long is made over the most prominent part of the tumour. (Fig. 44.) The superficial and deep fascial layers having been opened in the same line, and the depressor muscles of the hyoid revealed, these are cleared from the thyroid capsule and drawn aside. The capsule is then incised over the adenoma and the latter is shelled out by blunt dissection.

In the course of all goitre operations the veins, which are usually numerous and large, should be divided between ligatures as they are encountered.

In order to avoid having to open the wound for hæmorrhage after operation, it is a good surgical rule in all neck operations to let the patient come partially round from the anæsthetic, and to relax the tissues by raising the head, in order that all bleeding points may be seen and duly ligatured before the patient leaves the operating table.

A drainage tube should be inserted for twenty-four hours.

The wound is sutured in layers, the skin incision being accurately united preferably by the subcuticular suture with fine silk or silkworm gut.

(2) *Removal of a lobe for goitre* (partial thyroidectomy).—The lobe which is the larger is the one selected.

By means of the collar-incision, which in this type will be from four to six inches in length, and is lateralized to suit the more lateral operation, the superficial and deep layers of the cervical fascia are divided, exposing the depressors of the hyoid. (Figs. 44 and 45.) These muscles are then separated in the middle line, cleared underneath from the surface of the goitre and retracted or, if necessary, divided. Veins encountered are divided between ligatures. The outer fibrous capsule of the thyroid is now opened, and under it the finger is passed to effect the separation of the gland. Bands met with are not to be torn through, but are divided between ligatures. The muscles are strongly retracted, and the partly separated lobe which lies under the sterno-mastoid is pulled forwards, the upper cornu being cleared. Here are the superior thyroid vessels, and they also are divided between ligatures. The lower angle of the lobe is similarly dealt with so as to enable the inferior thyroid vessels to be ligatured, and this should be done close to, or actually within the gland substance, in order to avoid injury to the recurrent laryngeal nerve, which lies close to the artery—sometimes behind, sometimes in front of it. The isthmus is next cleared by division between ligatures of the vein-bearing fibrous tissue ; a director is insinuated between the trachea and the isthmus, and in its track one blade of stout pressure forceps is passed. The closure of these forceps crushes the thyroid tissue of the isthmus, and enables a strong ligature to be effectively tied round it.

The lobe is now all free save on its posterior surface, where it is adherent to the trachea and cricoid cartilage. A strip of the gland is usually left here to protect the recurrent laryngeal nerve, and the separation is effected with the knife, or by ligature and division. The subsequent course and after-treatment are the same as in the adenoma operation.

Difficulties and Dangers.—The operation is tedious rather than difficult. The dangers are first, hæmorrhage from cut or torn veins ; and secondly, injury to the recurrent laryngeal nerve.

Hæmorrhage can be reduced to a trifle, if the veins, which are numerous, large, and irregular, are isolated and ligatured in two places before being divided. This method is slow but safe, and is therefore preferable to that of the temporary application of pressure-forceps.

The recurrent laryngeal nerve is seldom seen during the operation, and yet paralysis of a vocal cord is common after operation. The explanation is that the paralysis is often due, not to division of the nerve at the operation, but to the nerve being stretched, and, above all, to its being involved in the subsequent scar-tissue.

The *Results of the Operation* are excellent, and after an aseptic convalescence the cicatrix is inconspicuous.

Exophthalmic Goitre (Graves' Disease) is supposed to be due to an excessive, and possibly perverted, secretion of the thyroid gland, the cause of which is unknown. The thyroid enlargement—unlike simple goitre—is accompanied by constitutional toxic phenomena, which serve to render a well-marked case unmistakable. These are:

1. *Eye Symptoms.*—Along with a variable amount of protrusion of the eyeball (exophthalmos) we generally find von Græfe's sign, which is elicited by making the patient look upward, and then slowly downward. If the upper eyelid delays in the descent of the eye, von Græfe's sign is present.

2. *Tachycardia*, the pulse-rate varying from 90 to 120 per minute when the patient is resting. Palpitation is easily evoked, and cardiac dilation may supervene.

3. Fine *tremors* of the muscles of the limbs and tongue.

Anæmia, dyspepsia, diarrhœa and emaciation are common in addition to the foregoing, and the patient is nervous, irritable, and easily excited. Milder forms of the disease, in which tachycardia and irritability of temper are the only signs, are by no means infrequent. Pressure-effects by the enlarged thyroid occur less frequently than in simple goitre.

Diagnosis.—The tachycardia, exophthalmos, and pulsation in the goitre differentiate Graves' disease from simple goitre. The combination of a raised pulse-rate with an enlarged thyroid should never be treated with thyroid feeding or with iodine externally.

Prognosis.—Graves' disease lasts for years, but generally tends to slow improvement and recovery, often with a dilated heart. The more severe cases may die of general asthenia and cardiac failure.

Treatment.—In its milder forms, exophthalmic goitre cna be controlled by rest and belladonna, with heart tonics. Exposure of the gland to the action of X-rays is advocated.

In the severer cases, the best and most successful treatment is the surgical removal of a lobe of the enlarged gland, and the operation should, if possible, be performed before the heart has begun to fail.

The operative technique is the same as has already been described, but the condition of the patient necessitates attention to several details of prime importance if we are to avoid post-operative death. Local anæsthesia by the infiltration of eucaine solution is preferred by many operators to general anæsthesia. If the latter is used, ether is preferred, and the patient is anæsthetized on several occasions prior to the actual day of operation, so as to deceive him as to the date of the operation, and to lessen nervous shock. The operation is performed as rapidly as possible, free handling of the gland being avoided, in order to prevent the absorption into the circulation of a dangerously large dose of the toxic thyroid secretion.

Acute thyroidism, which, when it appears, does so within the first twenty-four hours after operation, is denoted by a very rapid pulse, a rise in the temperature, and in the gravest cases by maniacal delirium. It is combated by morphine and atropin hypodermically. Acute thyroidism is occasionally encountered after operation on simple goitre.

The *Results* of operation in exophthalmic goitre, when attention is paid to the above points, are excellent.

Malignant Disease of the Thyroid Gland is usually sarcomatous in nature, but adenomatous cancer may occur. It generally attacks a thyroid already enlarged, grows with great rapidity, and disseminates widely all over the body.

The thyroid may be entirely removed for the disease, but chances of non-recurrence are extremely poor. In the meantime the patient develops myxœdema.

Acute Septic Thyroiditis with the usual phenomena of abscess formation is rare, and so is the interesting *acute* development of a *goitrous enlargement* usually but not invariably appearing in an endemic area.

Accessory Thyroids.—We have already alluded to the mesial lobe of the thyroid, the so-called *pyramidal lobe*, which lies near the middle line, and is attached to the upper border of the isthmus. The pyramidal lobe, which is by no means a rarity, being found in about one-third of all cases, is developed from the lower end of the thyro-glossal tract, which extends from the foramen cæcum of the tongue to the isthmus of the thyroid.

The pyramidal lobe shares in the enlargement in the different forms of goitre, and may itself require removal by operation. When enlarged it is palpable as a long, sausage-like tumour in front of the trachea and larynx, usually inclining to the right of the middle line. It is supplied by a special branch of the superior thyroid artery entering its upper pole.

The presence of a pyramidal lobe would render difficult the performance of a high tracheotomy, and if it can be felt prior to operation, should lead to the selection of the low operation. (See p. 181.)

Lingual Thyroid.—An accessory thyroid, which occasionally undergoes enlargement, is that found in the middle line of the base of the tongue below the foramen cæcum. When enlarged, it forms a smooth, rounded tumour projecting from the upper surface of the organ, visible in the laryngeal mirror, and sometimes also on simple depression of the tongue.

A hypertrophied lingual thyroid may cause difficulty in deglutition and a sensation of embarrassed respiration.

Treatment.—The tumour should be endured unless the thyroid in the neck can be plainly felt, as the lingual thyroid may be the only thyroid gland present, in which case its removal would be followed by myxœdema.

Operation.—If the cervical thyroid is present, the lingual thyroid may be removed by splitting the tongue in the middle line and shelling out the tumour from its base. (Stuart Low.)

Thyro-Lingual or Thyro-Glossal Cyst.—Along that section of the thyro-glossal tract which lies between the body of the hyoid bone and the upper border of the thyroid isthmus a cyst is liable to form if the tract fails to undergo complete involution. The cyst, which reaches the size of a pigeon's egg, is situated in or near the middle line of the front of the neck. From its upper border, the duct can usually be felt passing upwards towards the hyoid as a cord about the thickness of a lead pencil.

In the course of time, the thyro-glossal cyst may rupture and discharge through the skin forming the *thyro-glossal fistula*, the permanent orifice of which, exuding a scanty secretion, is visible as a puckered depression in the middle line of the neck.

The *only* treatment is by operation, and that is apt to be troublesome, as unless the entire duct is removed the cyst will re-form.

An incision is made in the middle line of the neck from the hyoid bone to the fistula, around which it loops, so as entirely to surround it. The duct is now carefully dissected out as far as the hyoid bone, behind which it disappears, merging into the periosteum of the posterior aspect of the body of the bone. In order to reach the extreme upper point, and to remove every trace of the epithelium-lined duct, the body of the hyoid is divided in the middle line with bone forceps, and the duct tissue behind it removed.

Then the two halves of the hyoid are united with stout catgut sutures, and the skin wound is closed.

TRACHEOTOMY; LARYNGOTOMY; AND INTUBATION

Tracheotomy consists in the opening of the trachea, and the insertion of a suitable cannula for respiration. It is "high," "median," or "low," according to whether the tracheal opening is made above the isthmus of the thyroid; in the situation of the isthmus; or below it. (See Fig. 46.) As a matter of fact, in the high operation it is usually necessary to detach the isthmus from the trachea, and to hold it down while the trachea is being incised, as the isthmus extends normally as high as the second tracheal ring, and in children may reach even higher.

The high operation is the operation of urgency; it is, therefore, the most frequently performed. Both the median and the low tracheotomy are operations of deliberation.

Indications.—Tracheotomy is indicated when asphyxia is threatened by any obstruction in the air-passages above the episternal notch. It is also performed when a loose, foreign body is known to be present in the trachea or bronchi, and for the administration of a general anæsthetic in certain operations on the larynx and pharynx.

Contra-Indications.—In phthisis pulmonalis, with tuberculous disease of the larynx causing stridor, tracheotomy may become necessary, but it should be postponed as long as possible, as its effect upon the lung disease is harmful.

Anæsthesia.—In adults, the operation is best performed under local anæsthesia, especially if there is any impediment to breathing in the larynx or pharynx. In children, general anæsthesia under chloroform is necessary.

But if the patient, whether adult or child, is in process of being asphyxiated, there is no time for anæsthetic of any kind. Moreover, the carbon dioxide poisoning dulls the sensibility to pain.

Operation.—The instruments required are: scalpels; dissecting forceps; pressure forceps; small retractors; sharp hook; dilators; and tracheotomy tube with pilot.

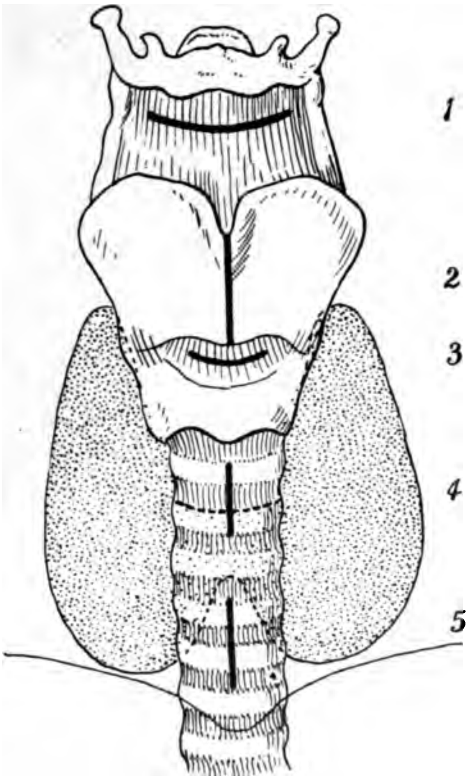


FIG. 46.—Larynx (Diagrammatic).

Incisions for (1) Subhyoid Pharyngotomy; (2) Thyrotomy (Laryngo-fissure); (3) Laryngotomy; (4) High Tracheotomy; (5) Low Tracheotomy.

Position.—The patient lies on his back with a pillow or sand-bag under the shoulders and the head as well extended as the breathing will permit of. In certain conditions, full extension of the head still further embarrasses the breathing. The operator stands on the right-hand side of the patient, an assistant steadies the head, keeping the middle point of the chin and the episternal notch exactly in line throughout the operation.

He may also administer the chloroform. If a second assistant is available, he takes his stand on the side opposite to the surgeon.

All instruments should be laid out before beginning the operation. The surgeon should operate under guidance of headlight if possible.

In the following description, it is assumed that the operation is deliberate. Emergency tactics will be described later.

Local Anæsthesia.—With a suitable syringe, the middle line of the neck from the cricoid to the sternum is infiltrated with 4 per cent. sol. of eucaine in water with a few drops of adrenalin in it. The needle is passed first immediately under the skin to its full extent, and then as it is being withdrawn, the solution is squeezed out. Two or three separate punctures may be required. Next the needle-point is passed more deeply into the fascial layers, the solution again being ejected as it is withdrawn. A third and a fourth series, still deeper, are usually necessary, and finally the needle point is plunged as deep as the trachea itself. Three to five minutes may be spent in gently massaging the injected area, and then the first incision may be made.

(1) **High Tracheotomy.**—The field having been prepared in the usual manner, the surgeon sees that everyone is in place, and all instruments to hand.

Then steadying the larynx between the fingers and thumb of the left hand, he makes an incision at least one-and-a-half inches long from the level of the cricoid cartilage downwards exactly in the middle line, dividing skin and superficial fascia from end to end in successive sweeps A few strokes of the knife brings him to the level of the sterno-hyoid and sterno-thyroid muscles, the interval between which is opened up by the blunt dissection. Now follow the one or two layers of fascia and areolar tissue that cover the trachea above the thyroid isthmus. These are penetrated by means of sharp cuts and blunt dissection, veins being avoided if possible, or divided between ligatures if time permit, otherwise they are cut between pressure-forceps.

The thyroid isthmus becomes visible as a soft reddish cushion. It is cleared from the trachea and pulled down. Incision into the thyroid should be avoided, as it is very vascular. A little more dissection brings us to the trachea, which should be exposed so thoroughly that its white rings are clearly seen.

Throughout this dissection, keep to the middle line. This rule is extraordinarily easy to transgress. If the assistant allows the chin to fall to one side; if the skin incision has deviated, even slightly; if a pair of artery forceps holding a vessel drags the wound to one side, the operator will go astray. The operator should retain his hold of the larynx throughout, and should keep feeling his way down to the trachea by constant palpation with the left forefinger.

But however plainly he *feels* the tracheal rings he should *not incise* them until he *sees* them.

The amount of trachea exposed should be rather more than is sufficient to accommodate the tracheotomy tube.

To resume: The trachea is now exposed. At this point, if time permit, puncture the trachea between two rings with a hypodermic needle, inject a few drops of 10 per cent. sol. cocaine within it, and wait five minutes for the anæsthetic effect. This obviates coughing after the tube is inserted. (For contra-indications to this step, see later.)

Next, the sharp hook, its point turned towards the patient's chin, is plunged into the trachea below the cricoid, and given to the assistant to hold. He holds it in such a way as to pull the trachea well forward into the wound, and is instructed on no account to remove the hook, or to let it go.

The surgeon then, with shortened scalpel, its edge towards the cricoid, stabs the trachea about the third ring, and carries the incision upwards as far as the sharp hook. The air can now be heard hissing into the trachea.

The dilator is next passed into the tracheal incision, opened and held open with the surgeon's left hand, while between its blades the tracheotomy tube, with its pilot in position, is passed. The pilot is withdrawn, and if the air is entering freely through the tube, the dilator is withdrawn. Finally, the surgeon takes the sharp hook out of his assistant's hand, and removes it himself from the trachea.

Ligatures are tied where pressure forceps are holding vessels; the shield of the tracheotomy tube is unscrewed and allowed to find its own level, then made secure again; and the gaping part of the skin-incision is sutured.

(2) **The Median Operation.**—The same incision, prolonged as far as the episternal notch, is employed. The early steps are the same as in the last operation, but when the isthmus of the thyroid is reached, it is cleared, and raised from the trachea by means of a grooved director passed under it. A couple of stout ligatures are passed under the isthmus, and tied in order that it may be divided between them. This done exposes the tracheal rings and the operation is completed as in the high operation.

(3) **Low Tracheotomy**—*Indications.*—The low operation is

preferred when the larynx is the seat of the disease, as of cancer or tubercle, since then it is important that the tracheotomy wound should be as remote as possible from the affected regions. The low operation is deeper, takes longer to perform, requires a longer tube, and there is apt to be more trouble during convalescence than with the higher operations.

Operation.—The incision extends from the cricoid to the sternum. A variable depth of cellular tissue and fat must be penetrated before the deeper layer of the cervical fascia and the sterno-hyoid and thyroid muscles are reached. Here those muscles of either side are in contact, and the interval between them must be precisely hit upon and opened up by blunt dissection. This gives access to another layer of cellular tissue, containing the inferior thyroid plexus of veins, which must be turned aside or divided between ligatures. Finally, the pretracheal layer of fascia is reached, and this being divided enables the surgeon to clear and expose the tracheal rings.

The subsequent steps for opening the windpipe and inserting the tube are the same as for the other operations, but a longer tube is required, or it will slip out of the trachea after the operation, and be a source of difficulty and danger until it is replaced.

Emergency Tracheotomy.—The above operations are described as they are performed under favourable conditions, with the patient placid and free from impending asphyxia, such as we find when a precautionary tracheotomy is performed.

In the presence of grave obstruction the operation becomes at once more difficult and more urgent, and with the time at our disposal so brief, deliberate dissection must be abandoned.

The high operation should be chosen, the trachea should be rapidly exposed by sharp dissection, bleeding points being quickly caught with pressure-forceps, or ignored, since when easy breathing is restored, the veins empty, and the bleeding stops of its own accord. Everything is sacrificed in order to open the trachea as speedily as possible.

One point only must be carefully watched. The *tracheal incision must not traverse the cricoid cartilage.* This precaution omitted, as it easily may be in children, there will be difficulty later on in getting rid of the tracheotomy tube. (See p. 164.)

In this operation there having been no time for an intratracheal injection of cocaine, a violent blast of coughing follows the insertion of the tube, and blood and mucus are blown up in the operator's face, unless he holds a pad of gauze over the tube. At the same moment, also, he must put his finger on the tube, or it may be coughed out of the tracheotomy opening. This bout is succeeded by a period of complete apnœa—physiological apnœa, due to the free inspirations that follow the introduction of the tube. It may be ignored.

In the **most urgent** cases, the trachea can be opened by *one stab of the knife* and without any anæsthetic, local or general.

The larynx is held as in the ordinary operation, but the fingers pressed in behind it pull it forward, thus rendering the skin tense. The blade of the knife is inserted straight through the skin, and down into the trachea, about the level of the first or second ring. As soon as it is felt to be in the trachea, cut carefully upwards for about half an inch. As the knife is withdrawn, press the finger into the trachea, and then insert the tube as the finger is withdrawn (Lambert Lack). Or, if a dilator is at hand, it may be inserted instead of the finger, opened, and the tube put in.

Hints in performing tracheotomy.

1. See that the head is well extended and held steady.
2. Let the light be the best procurable. Use a head-light or mirror if possible.
3. Do not turn aside for bleeding; it is the trachea you are after.
4. A long skin incision enormously facilitates progress, and increases safety.
5. Do **not** inject cocaine into the trachea in children, or if you wish the patient to cough, as in diphtheria or when there is suspicion of a foreign body loose in the trachea.
6. The sharp hook is the key to the position. Insert it well into the trachea; hold it, or have it held there until everything is finished and the patient quiet; then remove it yourself.
7. If bleeding continues from the wound after the operation, keep the patient anæsthetized, seize the deeper layers of the cut edges, and evert them without disturbing the tube until you find the bleeding point, which you then catch and ligature.
8. If blood is entering the tracheal wound, and setting up cough, and you cannot at once find the bleeding point, turn the patient over, and let the blood run out of the wound.
9. See that the trachea is opened in the middle line, and that the tracheal incision is long enough to receive the tube.

10. Hold the knife short in opening the trachea, so as not to wound its posterior wall.
11. Have the tracheotomy tube lubricated before trying to insert it.
12. Pass it through the expanded blades of a dilator if that instrument is at hand.
13. The insertion of the tube is often difficult. See that it gets into the trachea, and that the air is actually passing freely through it.
14. Do not remove the sharp hook until the patient is breathing comfortably through the tube.
15. Do not forget the inner tracheotomy tube.

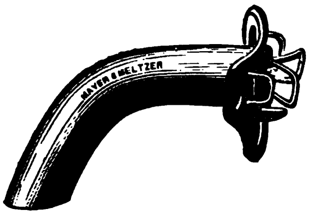


FIG. 47.—Parker's Tracheotomy Tube.

Tracheotomy Tubes.—Parker's (Fig. 47) is the best for children; Durham's lobster-tail (Fig. 48) for adults. A very long Durham (Fig. 49) is often necessary for low tracheotomy.

After the tube is inserted, and before the skin is sutured, the tube is secured by tapes tied round the neck.

After-Treatment.—A square of gauze, slit for the tube and introduced from below under the shield of the tube, is all that is required by way of dressing. No special tent or steam-kettle is necessary. A nurse must be at hand to wipe discharges away, and to keep the tube clear. This is done by means of a feather. The inner tube will need removing and cleaning from time to time.

The first day or two after the operation is a period of anxiety. If the patient is comfortable, the outer tube should on no account be taken out for cleaning for at least seven days. If it is, the layers of the neck close up, and the opening of the trachea may be lost, and with it the patient's life.

After seven days, a track is formed, lined with granulations. But even then, the changing of the tube must be carefully arranged. A second tube of the same pattern must be at hand to insert as soon as the first tube is removed, and with it dilators and forceps. All instruments must be laid on a table by the side of the bed, and *not on* or near the bed, for if the patient gets excited, he will move, and send them flying. Work, if possible, with a head-light, or with head-mirror and lamp.

If shortly after the operation the patient starts to cough, and continues coughing, probably the inner end of the tube is pressing on the tracheal wall and irritating it. Loosen the screw of the shield, and let it readjust itself.

If the coughing still continues, the patient may cough the tube out of the trachea, especially if it is a deep tracheotomy and the tube is rather short. Under such circumstances, the tube must be replaced by a longer one.

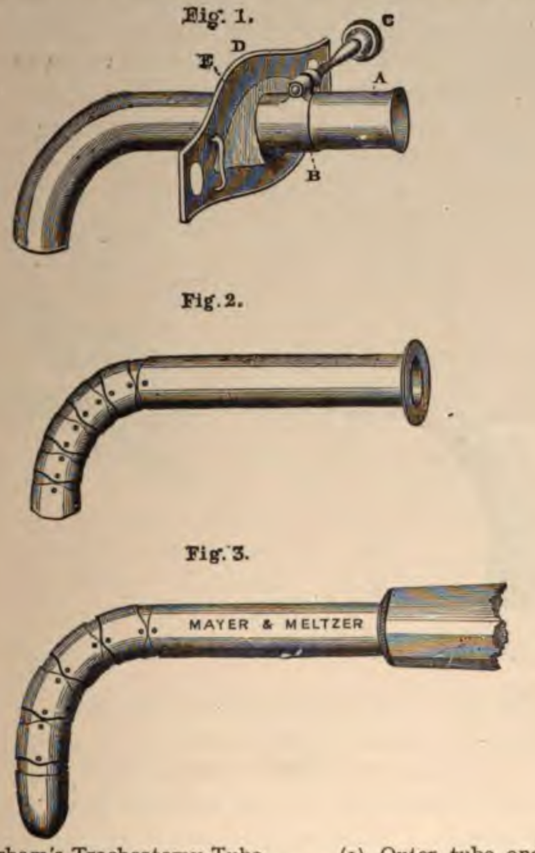


FIG. 48.—Durham's Tracheotomy Tube. (1) Outer tube and adjustable shield. (2) Inner tube (lobster-tail joint). (3) Pilot for introducing.

Prepare as before, but as soon as the first tube is removed, insert the dilator into the trachea, so as to keep the route open, and then insert the new tube through the blades of the dilator.

Coughing, if the tracheal wound is too large, and the skin is sutured, may induce emphysema. If this appears, remove one or two skin sutures. These manipulations may be facilitated, and the coughing soothed, by a little cocaine solution dropped into the trachea through the tube. But persistent coughing means an unsuitable or ill-adjusted tube, for which cocaine is useless.

Constriction of the trachea below the tracheal opening, as from a low goitre, may be overcome by the use of a long flexible tracheotomy tube, such as Kœnig's. As a temporary expedient, a length of drainage-tubing may be sufficient to permit of respiration being carried on.

The **Removal of the Tube** is undertaken as soon as the obstruction to the breathing has been got rid of. There is as a rule no difficulty in deciding when it may be done, as we can easily tell when the air is passing freely through the larynx again, and in any case this can be ascertained by trying whether the patient can breathe with the tracheotomy tube blocked.

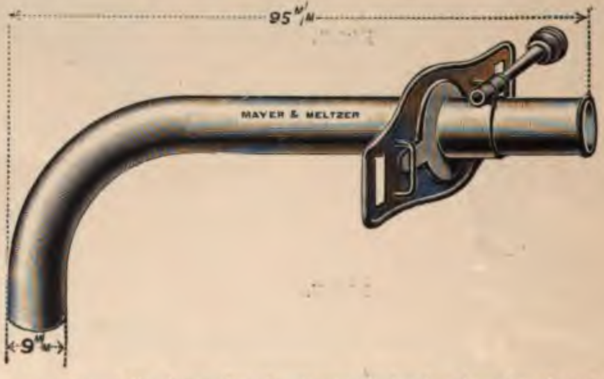


Fig. 49.—Specially long Tracheotomy Tube for low tracheotomy.

When he can do so, and there is no likelihood of a recurrence of the stenosis, the tube can be removed. A small pad of gauze and a strip of sticking-plaster across the neck so as to bring the edge of the tracheotomy opening together is all that is needed by way of dressing. The fistula closes readily.

Where the laryngeal or tracheal stenosis is permanent, the tube must also be worn permanently. In that case, the metal tube should be exchanged, after the track is formed, for a rubber tube, as the latter is much more comfortable to wear. This rubber tube should be removed, cleaned, and re-inserted daily, and as soon as it begins to show signs of wear, it should be replaced by a new one. These details the patient can attend to himself.

The wearing of a tube does not seem to have any harmful effect upon health; tracheotomy tubes have been worn for over fifty years on end.

Inability to remove the tube by reason of laryngeal swelling due to a tracheotomy which has been made too high, has already been discussed (p. 164). In addition to such obvious causes, it occasionally happens that a child fears the removal of the tube so much that asphyxia will be produced from sheer fright, or glottic spasm, if it is removed. This difficulty may be overcome by removing the tube under an anæsthetic, or by blocking it while the child is asleep.

LARYNGOTOMY

Description.—The opening into the larynx through the crico-thyroid membrane, as a temporary measure.

Indications.—Its most frequent use is in operations about the pharynx in adults, to permit of the more easy administration of the anæsthetic, and to prevent blood passing into the air-passages, since the upper part of the larynx can be packed after laryngotomy. But modern developments, such as the intra-tracheal administration of ether, have rendered laryngotomy less frequently necessary than it used to be. It is still occasionally performed, however, in place of an emergency tracheotomy in adults. In children, the space is too narrow to admit a tube.

The laryngotomy tube should not be worn longer than twenty-four hours.

Operation.—The patient is placed in the tracheotomy position.

The operation may be done under chloroform; local anæsthesia; or no anæsthesia at all.

A vertical fold of skin opposite the upper border of the cricoid cartilage is picked up, transfixed from side to side with a knife, and cut through so as to make a transverse incision about an inch in length. A pair of sharp-pointed scissors curved on the flat is plunged directly through the crico-thyroid membrane downwards and backwards in the middle line, keeping close to the upper border of the cricoid. The scissors are then opened wide, and the short, oval laryngotomy tube (Fig. 50), or a small Durham tracheotomy tube is inserted. See that the scissors are well into the air-passage, as one is apt to pass the tube between the crico-thyroid membrane and the tracheal mucosa.

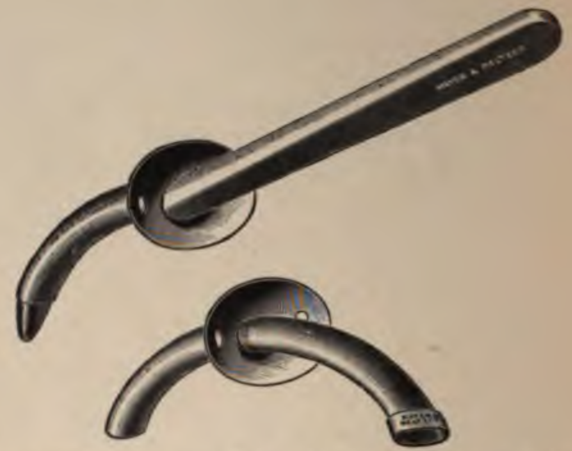


FIG. 50.—Laryngotomy Tube with Pilot and Inner Tube.

In an emergency, laryngotomy may be performed by a single stab of the knife through the crico-thyroid space, its place being taken by dressing-forceps inserted and opened.

After-Treatment.—The tube having been removed after operation, the wound heals up quickly.

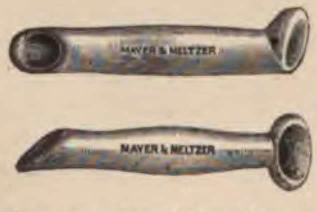


FIG. 51.—Intubation Tubes.

Intubation is sometimes preferred to tracheotomy for laryngeal diphtheria. It consists in passing into and leaving in the larynx an intubation tube of a size suitable to the patient's age. (See Fig. 51.) It is performed without an anæsthetic. The child is held upon the nurse's knee as described at p. 20. The mouth is propped open with a gag, inserted and held by an assistant standing beside the patient. The tube is threaded and held by the introducer, which the surgeon holds in his right hand, the thread being attached to his little finger. He passes his left forefinger into the throat, and with it hooks forward the small epiglottis. The introducer and tube, the latter with the beak turned posteriorly, are then passed into the mouth, the end of the tube passing under the tip of the left forefinger to enter the larynx, which it does in a direction downwards and forwards. The introducer is now detached. If properly inserted the dyspnœa is relieved at once. The thread is attached to the cheek with sticking-plaster.

Difficulties.—The chief difficulty is the finding of the laryngeal orifice in a struggling, semi-asphyxiated child, and even after it is found, one is apt to slip the tube past it into the œsophagus.

After it is inserted, a violent fit of coughing may eject the tube, and it has to be put in again. This can be prevented by using as large a tube as possible. And the use of a large tube also prevents its slipping down through the larynx into the trachea. Tracheotomy instruments should be at hand in case of need.

If, later, the tube is suddenly blocked, and the child is threatened with asphyxia, the nurse at once pulls it out. This accident seldom happens.

Removal of the Tube must be effected not later than five days after its insertion in diphtheria. It is pulled out by the thread, or if that has been removed, by the extractor. In little children, we can eject the tube from the larynx by compressing between thumb and finger the outside of the trachea, cricoid, and thyroid cartilages from below upwards—" milking the larynx," as it is called.

Intubation for Cicatricial Stenosis of the Larynx.—Intubation tubes made of vulcanite of graduated sizes are used, and being introduced, are worn for prolonged periods. By gradually increasing the calibre of the tube, the lumen of the larynx is gradually and permanently expanded until it is possible for the patient to respire through it.

FOREIGN BODIES IN THE TRACHEA AND BRONCHI

If large enough to block the lumen of the trachea, a foreign body will probably be arrested in the larynx, and cause instant death if not immediately removed therefrom. But if small enough to pass the guardian glottis, it may remain free in the trachea or, being aspirated into a bronchus, it may there become impacted. Foreign bodies are seldom arrested in the trachea.

If free to pass up and down the windpipe, violent expiration or coughing propels the foreign body every now and again against the under surface of the vocal cords, whereby glottic spasm is set up; or it may, by chance, become arrested in the chink of the glottis and cause death by asphyxia. Many of these cases concern young children from whom no history is obtainable, and thus whenever recurrent transitory attacks of glottic spasm, sudden and violent, occur in a child, we should always look for a foreign body, an X-ray examination being made as well as a direct examination of the trachea.

When a foreign body is fixed in a *bronchus*, the symptoms vary with the size and situation of the obstruction. If one or other of the main bronchi is blocked there will be dyspnœa with deficient expansion of one lung, and frequently pain in the chest. If the foreign body finds its way into one or other of the smaller bronchi, the symptoms become more obscure; dyspnœa is not a prominent symptom, although pain may be complained of. In a large number of cases, the presence of a foreign body remains unsuspected, the only symptoms being such as commonly arise from pulmonary disease—cough, expectoration, and the formation of cavities, or bronchiectasis. In the last-named particularly, the pathogenesis of which is seldom quite clear, the possibility of foreign body should always be excluded by examination.

Many patients with a foreign body in the chest can give no history of having inspired or "swallowed" a foreign body, but experience has proved that when a patient does give us such a history, we should invariably accept his word for it and examine accordingly.

The effect of a foreign body impacted in a bronchus varies also according to its nature. Small objects of metal, glass, and undecomposable material generally may be tolerated without any secondary evil results for months or years. Such results are always to be looked for, however, sooner or later.

In the case of bodies such as peas and beans, on the other hand, which, being soft and friable, decompose readily, break up into small pieces, and become disseminated, the effects are much more prompt in appearing. They are also more dangerous, seeing that they consist of acute pulmonary infection of the bronchitic, pneumonic, or even gangrenous type.

Diagnosis.—A history of foreign body having been obtained, every effort must be made to locate its situation, or, on the other hand, to show that it is not present.

The history may also indicate the character of the body. If metallic, a skiagram should reveal its presence and its position. It is important, especially when dealing with a fine body such as a pin, that the picture taken be instantaneous, otherwise its movements in respiration will prevent it appearing on the negative. Radiologists have become highly expert in locating precisely the situation of bodies in the chest, and it is, moreover, frequently advantageous, as Tilley first pointed out, to make our search for the body under the guidance of X-ray illumination and the screen.

If the history indicates that the foreign body is of some such character as a bean, or a small piece of nut-shell, which is permeable to X-rays, no time should be spent in radiography, but, especially if acute pulmonary symptoms have set in, immediate search should be made with the direct tube.

Treatment.—(1) *A foreign body loose in the trachea* necessitates immediate tracheotomy, as the patient is in instant peril of asphyxia from glottic spasm or impaction of the body in the larynx.

A high tracheotomy is performed, cocaine not being injected into the trachea, and after it is opened, the edges of the tracheal incision are held apart to permit of free airway for the expulsive cough, as with that cough the foreign body is generally ejected.

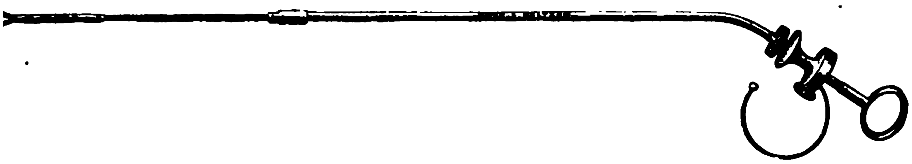


FIG. 52.—Brüning's Bronchoscopy Forceps for Foreign Bodies.

(2) *A metallic body impacted in trachea or bronchus.*—The position having been located by symptoms, and by X-ray examination, a bronchoscope is passed down as near to the foreign body as can be accomplished, and then with suitable forceps it is seized and withdrawn.

The forceps for this purpose at our command are many and various (Fig. 53), and the kind selected will depend, to a great extent, upon the character of the body. The removal of pins, needles, and pieces of blunt metal is a comparatively simple problem. Beads and similar hard, slippery objects are naturally more difficult to secure, but can sometimes be caught and held by expansile forceps passed closed into their interior, and then opened.

After the foreign body has been caught, the patient should be inverted somewhat, so that if our hold is lost, the foreign body falls out through the tube, and does not drop back again into the bronchi.

In infants and little children, only the smallest possible bronchoscope should be used by upper bronchoscopy (i.e., the tube being passed through the larynx) and the *séance* should not exceed fifteen minutes. Otherwise, glottic œdema will follo and a tracheotomy be required. Indeed, many experts pre in little children to perform tracheotomy first of all, and th to make the search, passing the bronchoscope through t tracheotomy opening (lower bronchoscopy).

(3) A soft, friable body may be located by the distributi of the lung symptoms, but if the reaction has been acu or widespread, this may be difficult or impossible, in whi case the search will have to be made in both main bronch systems. We may be visually guided to the site by lo signs of inflammation.

The dangerous nature of such objects renders their immedia removal important, and every effort must be made to extr them entire. For this reason, no time should be wasted w upper bronchoscopy; tracheotomy ought straightway to performed, and lower bronchoscopy adopted. The same rules to be followed as described above, but the forceps used m grasp the body softly, so as not to break it or crush it i fragments.

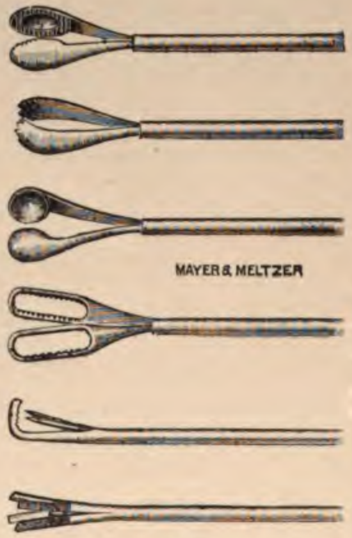


FIG. 53.—Various Bronchoscopic Forceps for Foreign Bodies.

Granulations may have sprouted up on the proxim side of the foreign body, and may conceal it. Applicati of adrenalin solution will cause them to shrivel, otherwise t forceps must be passed through them, and the foreign bo groped for.

It is sometimes possible in the case of round slippery bodies to pass the end of the tube close on to the body, and then to make a pad of wool, soaked in oil or vaseline on a long swab-holder, act in the tube as a piston, aspirating the foreign body against or into the tube, so accomplishing its removal.

When the body is too long or large to enter the tube, the forceps are made to retain their grasp, and tube, forceps and foreign body are all removed together. This manœuvre is obviously unsuited to sharp or pointed bodies, but such bodies can generally be manipulated so as to enter the tube.

Search for a foreign body in a case of bronchiectasis is a difficult manœuvre, as the pus is both copious and fœtid, even when this factor is minimized by keeping the patient inverted for a day or two before the operation.

Since the introduction of direct bronchoscopy, the success in extracting foreign bodies from the bronchi has been very striking, and many lives have, in consequence, been saved thereby. At the same time it is well to recognize that there are cases in which a foreign body in the lungs cannot be extracted by this method. For such of these as require removal, the only remedy is removal by external operation through the chest-wall (pneumonotomy).

(For the extraction of objects like safety-pins, see p. 211.)

THE ŒSOPHAGUS

The following are some anatomical facts of clinical importance regarding the œsophagus:

The length of the food passage from the incisor teeth to the gastric orifice is 14 to 16 inches. The laryngo-pharynx narrows to become the upper end of the œsophagus opposite the cricoid cartilage and the sixth cervical vertebra. The lower end of the gullet pierces the diaphragm opposite the tenth dorsal vertebra and enters the stomach opposite the eleventh.

There are three constrictions—one at the commencement, the other, very slight, opposite the aortic arch or bifurcation of the trachea (about 13 inches from the incisors), and the third where the gullet pierces the diaphragm. Strictures of the œsophagus, both malignant and cicatricial, are commonest at these constrictions, and foreign bodies are most readily arrested at these points.

The upper, or post-cricoid constriction belongs, strictly spe ing, to the pharynx and not to the œsophagus, seeing that t muscular fibres are the lower fibres of the inferior constrict pharyngis, which are here strengthened to form a sphincte But for our present purposes, we shall consider this regio along with the œsophagus, except in so far as post cricoid cancer is concerned, that having already been dealt with. (See p. 66.)

ŒSOPHAGOSCOPY

The direct telescopic tubes used for the trachea and bronchi may also be used for the examination of the œsophagus, but there are many other varieties at our disposal. The length and calibre are dictated by the age of the patient, but it is a safe rule to use a wide tube when searching for a foreign body, and a narrow tube for ordinary examination, and especially when malignant disease is suspected.

The passage of the tube into the œsophagus is much more difficult, and it is also more dangerous than tracheoscopy, but attention to the following directions will minimize both difficulties and dangers alike, particularly if the surgeon is careful to avoid force and haste.

Anæsthetization.—Local anæsthesia, when it can be satisfactorily accomplished, is safer than general anæsthesia. It also permits of the sitting position, which has many advantages over the recumbent.

In children, however, a general anæsthetic will usually be necessary.

In inducing local anæsthesia for œsophagoscopy, the pharynx and larynx are anæsthetized as described at p. 92. In addition, by means of long bent swab-holders, the laryngo-pharynx is reached by way of the pyriform fossæ. As the œsophagus proper is devoid of acute sensibility we do not require to anæsthetize it.

Before the sitting, the patient should receive a dose of morphia and atropin hypodermically (gr. $\frac{1}{4}$ morph. and gr. $\frac{1}{100}$ atropin sulph.) to calm apprehension, and reduce salivary secretion.

Method.—The passage of the instrument as far as the introitus laryngis is as in direct laryngoscopy.

The left arytenoid eminence having been recognized, the end of the tube glides past, not over it, into the left pyriform sinus, while at the same time the proximal end of the tube is borne rather backwards and towards the right angle of the mouth. After entering the pyriform sinus, the tube is arrested for a longer or shorter time by spasm of the post-cricoid sphincter, and as all that is visible through the tube at this place is smooth mucosa, without pucker or break, the operator is apt to think his direction is false. After a minute or two of waiting, the end of the tube should be pressed forward, so as to carry the cricoid cartilage in an anterior direction. At the same time the tube is pushed on downwards, but not forcibly. Then quite suddenly the œsophageal orifice will gape, and a little further advance brings the end of the tube into the œsophagus proper, which in normal cases is now open and visible as far as the lower thoracic section as a pale, smooth-walled, gently-curved tube, against the left side of which the heart can be seen beating.

The œsophagoscope, correctly directed, now passes on without any difficulty, if the œsophagus is healthy, to just above the diaphragmatic constriction, at which level the patient's head, hitherto held slightly forward and deviated slightly without rotation to the right so as to bring the end of the tube into the correct position for the lower œsophagus, should now be more fully extended. The diaphragmatic constriction is more easily negotiated than the cricoid sphincter, and then, the short abdominal œsophagus having been traversed, the end of the tube enters the stomach.

The withdrawal of the instrument should be very slowly effected, and under inspection throughout, as it is often possible to make a more searching and deliberate scrutiny of the mucosa during withdrawal than during insertion.

When the œsophagus is the seat of disease or a foreign body, it usually does not manifest itself as an open canal after the tube has passed the cricoid level, but remains closed as far as the obstruction. When, therefore, after passing the cricoid sphincter the œsophagus fails to open out in front of the tube, we should take that as a warning to be cautious in our further efforts.

Dangers.—The wall of the œsophagus is delicate, and is easily torn, especially when it is infiltrated with cancer, and a rupture almost invariably leads to septic mediastinitis and death. Rupture is also easy to produce in cases of pouching, the end of the œsophagoscope being inadvertently pushed through the *cul de sac* of the pouch. If the accident is recognized at the moment, and if the tear is situated at or slightly below the cricoid, the neck should at once be opened, under a general anæsthetic, in order to expose and drain the seat of injury. If, on the other hand, the injury affects the thoracic or abdominal œsophagus, it is beyond repair.

These facts emphasize the teaching of gentleness and patience in practising œsophagoscopy. (See also under Cicatricial Stricture, p. 204.)

Sounding the Œsophagus.—In addition to œsophagoscopy, we can frequently obtain some information by passing an œsophageal bougie.

When suspicion of stricture is aroused (when the history tells of the arrest and maybe regurgitation of food from the gullet after swallowing), the œsophageal bougie may be used to ascertain whether or not the calibre of the tube is organically narrowed. A bougie 14 millimetres in diameter should pass easily, otherwise there is stenosis. If, when the bougie is *in situ* in cases of stenosis, the point on the instrument corresponding to the incisor teeth be marked, and the distance of this point from the end of the bougie measured after withdrawal, the approximate site of the constriction in the gullet will be discovered.

In passing the bougie, after having smeared it with glycerine or vaseline and bent it slightly near the end, one carries the point of the instrument to the back of the pharynx, and, under the guidance of the left finger in the pharynx, pushes it down, and rather to the left with a steady, sweeping movement. Arrest by muscular contraction is common at the upper constriction, but the spasm soon passes away if no attempt be made to force the instrument onward. On withdrawing the instrument, note if there is blood or other discharge on the tip.

The bougie being formed of gum-elastic, is less apt to damage the œsophageal wall than is the rigid tube. But *it should never be used if there is* any suspicion of a *foreign body* in the œsophagus. It is advisable also to have the patient X-rayed before proceeding to examine the œsophagus by any of the above methods, save, perhaps, in recent cases of foreign body, where time is of value.

AFFECTIONS OF THE ŒSOPHAGUS

Malformations.—Congenital communication between the œsophagus and trachea is occasionally found. Its presence is, of course, incompatible with life, and children born with the deformity die in a few weeks.

Acquired Malformations.—Diverticula are of two varieties:

(1) Pressure diverticula.
(2) Traction diverticula.

1. **Pressure Diverticulum, Œsophageal** or **Pharyngeal Pouch** (Fig. 54) is found springing from the posterior wall at the junction of the pharynx with the œsophagus. It seems to be due to a congenital defect or weakness in the muscular coat of the lower pharynx. As a result of the weakness in the wall, a pouch is gradually formed lying between the œsophagus and the vertebral column. Thus, a pouch is in reality a hernia of the lower pharynx, through or under the fibres of the inferior constrictor muscle.

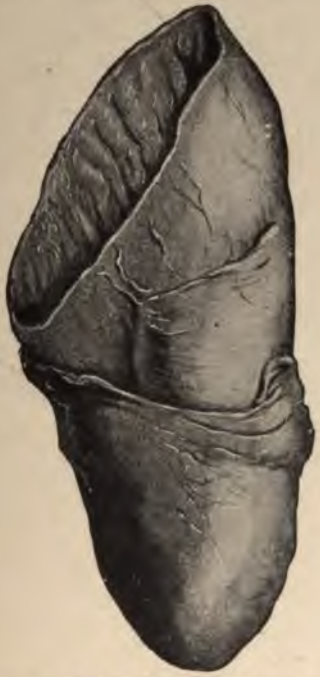


FIG. 54.—Pharyngeal Pouch (removed by W. H. Kelson).

Etiology.—According to one view, the weakness is congenital, as it is in abdominal hernia. According to another, the pouch is produced by the mechanical pressure of food imperfectly masticated and hurriedly swallowed. Pouching is commonest in males and rarely gives rise to trouble before the age of thirty.

Symptoms.—Food passes into the pouch, and is regurgitated

into the mouth some little time after it has been swallowe
On examining with the laryngeal mirror, and exercising, at t
same time, pressure over the upper part of the neck, food an
saliva may be seen pouring out of the slit-like orifice of the pouc

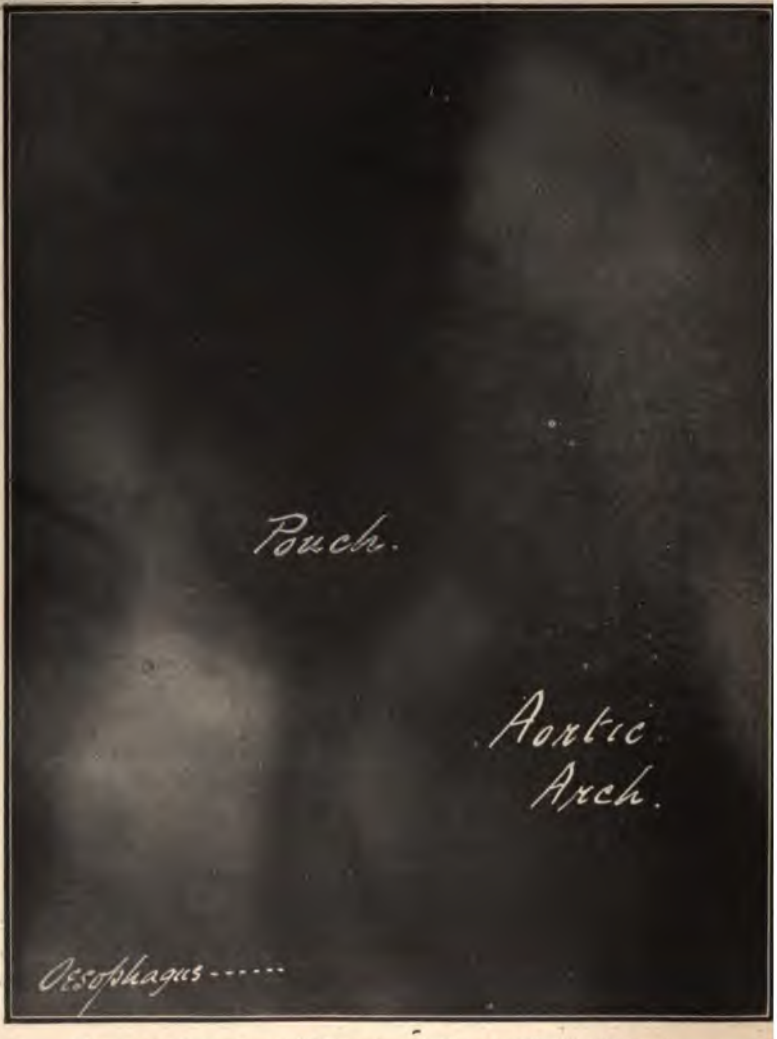


FIG. 55.—Skiagram of Pharyngeal Pouch (W. H. Kelson).

When a bougie is passed, it will generally become engaged an
arrested in the diverticulum.

Under examination by the X-rays and the screen, and b
getting the patient to swallow some water containing bismut
or barium, the shadow may be seen passing into the pouch an

stopping short in the neck instead of disappearing into the thoracic cavity as it does when the bismuth passes down into the normal œsophagus. (Fig. 55.)

Examination by the direct tube is difficult, as the posterior pharyngeal wall leads directly into the sac, and if any force is used, the tube will tear through the thin membranous fundus.

The *Diagnosis* may be made by observing the emptying of the sac from outside pressure. Otherwise, the symptoms are very similar to those of stricture, and the diagnosis may be dependent entirely upon the examination by X-rays.

Treatment.—The patient soon learns to empty the pouch by pressure from without after meals. It may be removed by operation. An incision is made along the anterior border of the left sterno-mastoid with its centre opposite the cricoid cartilage, and access to the pouch obtained by dissection down to the inner side of the carotid sheath, in the course of which the omo-hyoid muscle and the superior thyroid vessels must be divided. The pouch is cut off level with the œsophago-pharyngeal wall, the opening being carefully stitched, and the wound in the neck drained.

The results are generally satisfactory, but the pouch may recur.

2. **Traction Diverticula,** which are very rare, are due to cicatricial adhesion of the wall of the œsophagus to a bronchial gland, and so occur about the bifurcation of the trachea. They are seldom recognized during life.

Œsophagitis.—Inflammation of the œsophagus is due to the swallowing of corrosive or irritant liquids. It may also follow injury by foreign body. It is associated with severe pain; dysphagia; and, if the œsophageal wall is penetrated, with mediastinitis or cervical cellulitis and collapse.

Treatment.—The corrosive liquid should be neutralized. Thus acids are counteracted with weak alkalis, such as bicarbonate of soda, and caustic alkalis with dilute vinegar. In both, oil may be given.

Local **ulceration** of the œsophagus may result from impacted foreign bodies, or from such diseases as epithelioma. (See p. 207.)

Varix of the veins of the lower end of the œsophagus, an occasional source of fatal hæmatemesis, is found in cirrhosis of the liver.

At this situation also occurs the rare **peptic ulcer** of the œsophagus, which, like gastric or duodenal ulcer, has a tendency to perforate. It is commoner in males than females, and like duodenal ulcer, is a disease of middle life.

The *symptoms* are pain at the lower end of the sternum and between the shoulders, with vomiting and sometimes dysphagia from reflex œsophageal spasm, a feature which distinguishes it from peptic ulcer of the stomach.

Treatment.—The ulcer is treated by the local application through the œsophagoscope of silver nitrate, bismuth, or other astringents.

STRICTURE OF THE ŒSOPHAGUS

Stricture of the œsophagus may be due to disease of the œsophagus, or to the pressure of tumours, goitre, aneurysm, etc., situated external to the œsophagus. There are three varieties of the former :

1. Spasmodic stricture ; and
2. Organic stricture, which in turn is divided into
   - (a) Fibrous, and
   - (b) Malignant stricture.

Spasmodic Stricture, although perhaps most common in women, affects also men, and it may occur—indeed it frequently makes its first appearance in infancy. And in addition to spasmodic stricture from some neurotic cause, spasm nearly always accompanies irritation of the œsophagus, as from inflammation, ulceration, malignant disease, or a foreign body. For that reason, as Hill has pointed out, we should be careful to exclude organic disease before diagnosing the case as one of functional stricture.

Three varieties are described according to the location of the stricture.

1. *Post-Cricoid Stricture,* which is, in reality, a spasm of the lower fibres of the inferior constrictor of the pharynx rather than a spasm of the œsophagus, induces a difficulty in swallowing which the patient usually refers to the throat. In cases seen by the writer, the difficulty attended every meal, and frequently necessitated rising from table to regurgitate. It may last for many years, but the patient is usually able to get enough nourishment down to prevent serious emaciation.

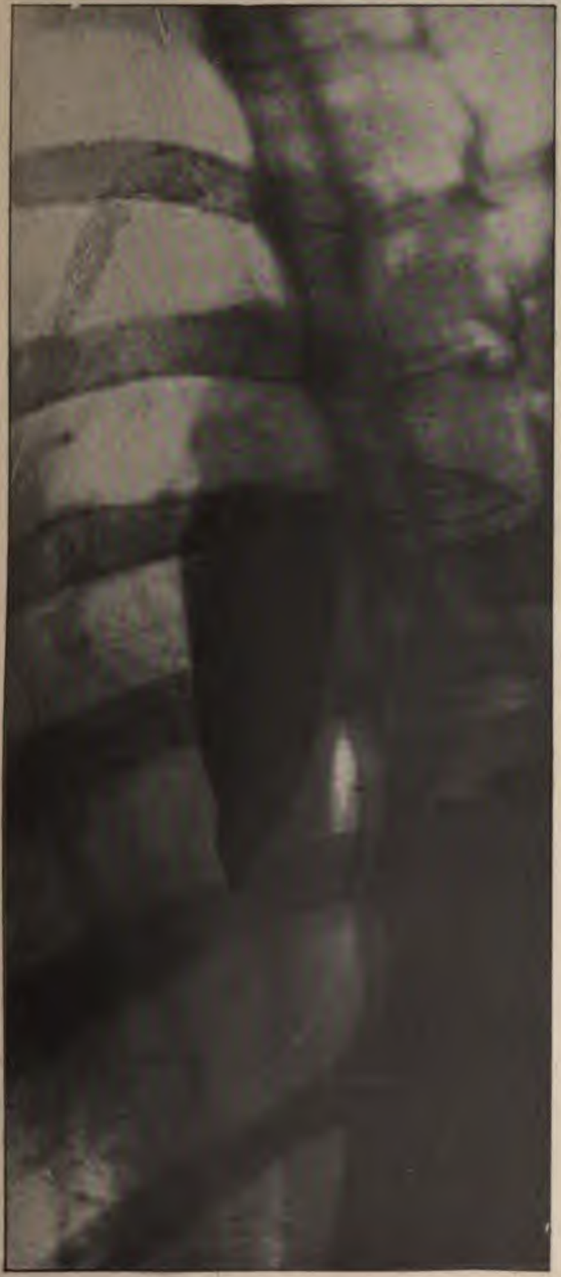


FIG. 56.—Spasmodic Stricture of Œsophagus in mid-thorax. Cured by suggestion. (Skiagram by Dr. Robert Knox.)

Œsophagoscopy shows the post-cricoid or pharyngo-œsophageal lumen reduced to a diameter of 3 to 5 mm., and looking as if a thread were tied under the mucosa. Examination with X-rays and screen shows the bismuth meal delayed in the post-cricoid region.

Treatment by passage of the bougie through the œsophageal tube is followed by immediate cure, but the stricture may recur from time to time, and require another passage of the sound.

3. *Intra-thoracic Spasmodic Stricture* causes similar symptoms, but the stricture varies from time to time in tightness, coming and going capriciously, sometimes being so tight as to arrest even liquids, at other times being quite absent. (Fig. 56.)

The bougie also leads to the cure of this variety.

4. *Spasmodic Stricture of the Cardiac End of the Œsophagus : (Cardio-spasm, Achalasia of the Cardia.)*—The name "achalasia," meaning the absence of dilatation, is probably the best expression, since the condition seems to be due not to spasm, but to paresis or paralysis of the vagus. When that nerve is stimulated, the œsophagus above the cardiac end undergoes contraction, while the cardiac sphincter dilates, this being the normal movement of the œsophageal wall. When, on the other hand, the vagus is experimentally divided, the œsophagus, as a whole, dilates, while the cardiac end remains tightly closed.

This is the state of matters in the condition we are discussing, which is the most serious of the neuroses of the œsophagus.

(We must add that the above explanation of muscular spasmodic obstruction of the lower end of the gullet is not universally accepted.)

Here, as in the last form, the obstruction may come and go, but it sometimes lasts for weeks, and even for years, leading at last, after many ups and downs, to serious emaciation, asthenia, and even to death.

The dilatation of the thoracic œsophagus above the cardiac sphincter may reach enormous dimensions, rivalling in capacity the stomach itself (Fig. 56), and as time passes, the stricture, functional at first, tends, by the onset of chronic inflammatory change, to become organic.

Symptoms.—Partial obstruction, when semi-solids or liquids are able to pass, alternates with absolute obstruction, due often to the impaction of imperfectly chewed pieces of meats, etc.

The dilated œsophagus holds large quantities of food and liquid which the patient learns to regurgitate. It is advisable to test this regurgitated food for hydrochloric acid in order to make sure that the case is not one of pyloric obstruction and dilatation of the stomach.

The condition may be extremely chronic. One case reported by the writer had lasted sixteen years when he was first seen.

X-ray examination should always be employed. It reveals the site of obstruction to be at the cardia, and will show if the lower œsophagus is dilated. (Fig. 57.)

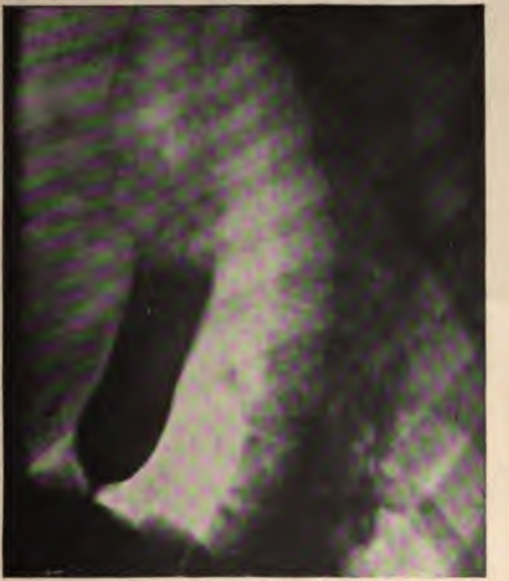


FIG. 57.—Stricture of the cardiac end (achalasia) of the œsophagus, with dilatation above. (Skiagram by Dr. Robt. Knox.)

On examination with the œsophagoscope during the phase of total obstruction, the direct tube plunges into a well of dirty liquid, containing, it may be, pieces of solid meat.

Treatment.—This being emptied by means of the aspirator, or by inverting the patient while the direct tube is in the œsophagus, the cardiac orifice (or it may be the hiatus) is revealed, and can be threaded first with a fine bougie, and then gradually with larger sizes. The largest size should be permitted to remain *in situ* for two or three hours at the first sitting.

The treatment is not free from danger. In these circumstances the œsophageal wall is friable, the orifice may be difficult to find, and if the bougie is incautiously pushed on, it may tear through the wall of the œsophagus.

If difficulty is experienced, an attempt should be made to pass Hill's feeding tube (see Fig. 58) through into the stomach, and to maintain nutrition by its means. This failing, gastrostomy should be performed, and a second attempt made later.

Sometimes a general anæsthetic relieves the contraction at the cardiac end.

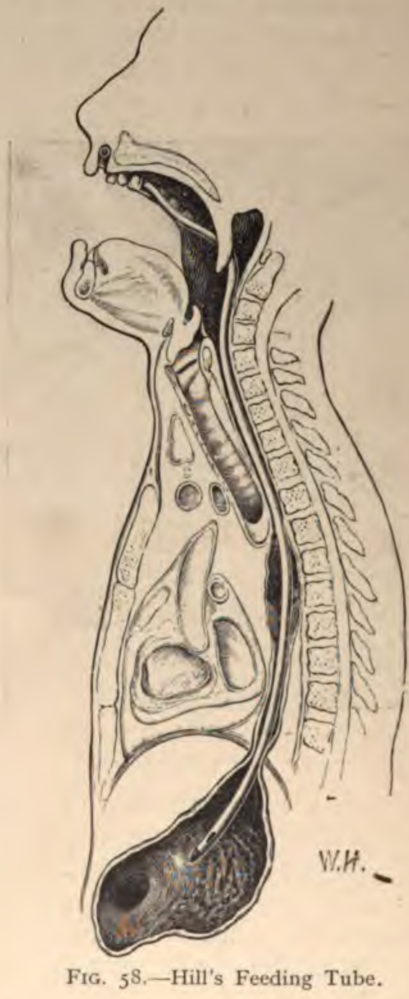
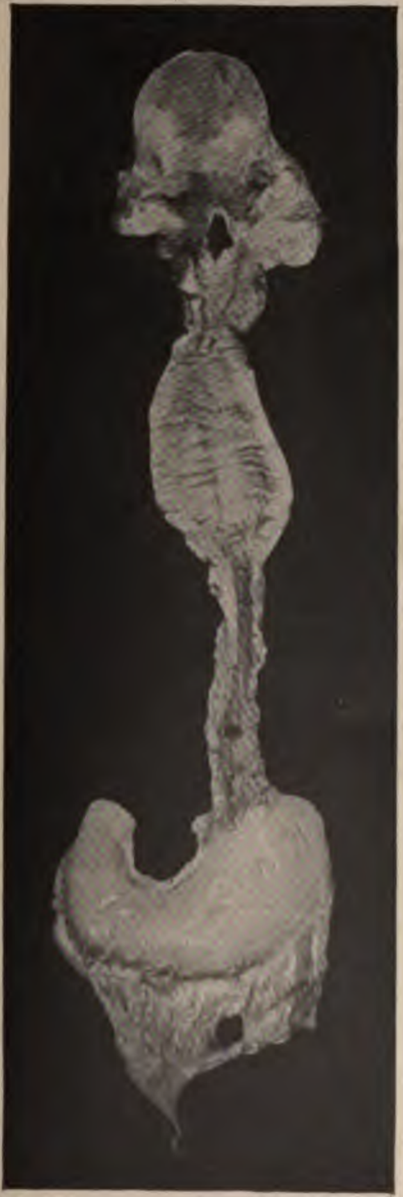


FIG. 58.—Hill's Feeding Tube.

Cicatricial Stricture (Fibrous Stricture) may form in any part of the gullet, and there is some divergence of opinion as to its commonest situation. (See Fig. 59.) The swallowing of a corrosive poison is perhaps the most frequent cause, but it may also result from the ulceration produced by a foreign body; from syphilitic disease; and from the abrasion and the ulceration ent upon spasmodic stricture. Finally, a fibrous stricture m without any obvious cause.

ptoms.—The symptoms arising from fibrous stricture tend



Tongue, larynx, œsophagus, and stomach of an infant, showing stricture of the œsophagus induced by a corrosive poison. Note the dilatation of the œsophagus above the stricture.

to become progressive, and to lead to absolute obstruction and death. The history is that of difficulty in the swallowing, first of solids, and later of liquids. Periodical attacks of absolute obstruction from the impaction of unchewed food or of substances such as pills or tabloids, are common, but the patient soon learns to relieve himself of such attacks when they set in, by a movement of regurgitation or by the induction of vomiting.

As in achalasia of the cardia, the œsophagus above the stricture undergoes dilatation, and in this sac the food collects, regurgitating after a time. Attacks of inflammation and ulceration are common, as in the cicatricial strictures of other canals, and may also lead to absolute obstruction.

When *absolute obstruction of the œsophagus* is set up, from whatever cause arising, the immediate peril to the patient arises less from want of nourishment than from want of liquid—the so-called "*water-hunger.*" In this state, which appears in from three to six days after the onset of complete blockage, the patient is in a dangerously weak condition, and all examination should be suspended until the urgency is relieved. In order to do so, gastrostomy should at once be performed, and water given by the artificial opening. In the meantime, normal saline solution should be given per rectum and subcutaneously, but these measures should be considered as supplementary only to the gastrostomy.

When the water-hunger is relieved, and not till then, the œsophagoscope may be passed, and the œsophagus examined.

The first task may be to empty the dilatation above the stricture of the food, solid and liquid, within it. This can be done by means of a long aspirator passed into the dilatation through the direct tube, or by inverting the patient while the tube is in the œsophagus. The stricture then becomes visible as a puckered or dimpled point, around which the mucosa is often red, swollen, and ulcerated, bleeding readily when touched.

Diagnosis.—If the stricture be situated at the cardiac end it can only be distinguished from achalasia by the results of treatment. The latter when simple is at once cured by dilatation, while the former is much more obstinate.

Compression of the gullet from without is distinguished from stricture by its being linear or crescentic, whereas the stricture shows as a puckered orifice. The mucosa also in compression is smooth and uncongested.

From malignant disease fibrous stricture is usually easily distinguished on œsophagoscopic examination by its smoother upper orifice, and by the absence of fungating masses.

Prognosis.—Cicatricial stricture of the œsophagus tends to become absolute, and to cause death, but the results of treatment are good if the patient can be brought under control before the onset of water-hunger, that state being one of immediate peril.

Treatment.—Having brought the patient within the limits of safety as directed above, the next step is to give the œsophagus rest so as to reduce local swelling and inflammation. Nothing is given by the mouth save a little bismuth, and the patient is kept in bed.

In a few days we may proceed to dilate up the stricture by means of bougies. If it is very fine, Chevalier Jackson's filiform bougies are used, being passed through the stricture by gentle continuous pressure. Previous to the operation, the patient swallows some olive oil as a lubricant. Once passed the bougie is allowed to remain *in situ* for half-an-hour. A few days later a series of larger bougies is passed, and soon the patient is able to swallow well-chewed food.

The gastrostomy opening may then be closed. But the patient should be examined once a month, and every indication of tightening of the stricture met by timely treatment. At home, the patient himself may keep the lumen from closing by passing a bougie from time to time.

The most difficult and troublesome strictures are those which are multiple, and much patience is required for their negotiation.

Impermeable Stricture of the cervical œsophagus may be treated by œsophagotomy, excision, and the turning in of a skin-flap as in œsophagotomy for cancer. (See p. 68.) If the impermeable stricture is situated in the thoracic œsophagus, gastrostomy is the only available measure. If it is situated in the lower third, it may be divided through a gastrostomy opening by the surgeon cutting on to the end of the œsophagoscope passed from above, the lumen being maintained until healed by tubes, and later by regular bouginage. (Chevalier Jackson.)

CANCER OF THE ŒSOPHAGUS

(Malignant Stricture of the Œsophagus.)

(For post-cricoid cancer, see p. 66.)

Cancer of the œsophagus may commence at the pharyngeal end, at the middle, or at the gastric end. The growth, which is epitheliomatous, grows round to involve the whole circumference of the tube and so induces stricture.

It causes death by water-hunger; by general asthenia; or by extension to neighbouring structures and organs, such as the large blood-vessels, the trachea, or the pleural cavities.

Symptoms.—The patients are usually men over forty years. The first symptom to appear is usually difficulty in swallowing, and although of gradual onset as a rule, the earliest intimation of the presence of the disease may be the sudden onset of complete obstruction. When progress is more gradual, the dysphagia is associated with progressive emaciation. Cough after attempted swallowing, with, sometimes, pain referred to the seat of the disease, is frequently present.

Enlarged cervical glands may be palpable at the root of the neck, and they are sometimes found before any suspicion has arisen that the disease is œsophageal, as when, for example, a patient comes complaining of hoarseness which laryngoscopic examination shows to be due to recurrent paralysis. In such a case the presence of hard, enlarged glands at the root of the neck would indicate cancer of the œsophagus.

If there is regurgitation of food after swallowing, the presence of blood in the regurgitated matter would also raise suspicion of cancer.

The symptoms of the terminal stages depend upon the nature of the treatment, and on the direction taken by the infiltrating disease.

Examination.—Before examination with the direct tube is undertaken, an X-ray examination with bismuth should be made, in order to determine the exact level of the obstruction.

Œsophagoscopy should only be undertaken with the precautions outlined above (see p. 194), and no attempt should ever be made to force the tube through the stricture, as the cancerous œsophageal wall is extremely friable.

The appearances are those of stricture, with infiltration, ulceration, and fungation of the mucosa. Gentle pressure by the end of the tube is sufficient to cause bleeding, and the bright-red masses of cancerous fungations are, as a rule, sufficiently distinctive. If necessary, a portion may be removed for microscopic examination.

Treatment.—The only method we can adopt for carcinoma lower than the cricoid level is to apply *radium*, and, as experience shows, this is only a palliative remedy.

Method.—A suitably screened tube containing 50 to 100 mgm. of radium bromide, or its equivalent in radium eman attached to a flexible wire, is passed through the œsophagoscope into the lumen of the gullet at the seat of the growth, and left there from eight to twelve hours. The patient, meantime, is kept in bed, under morphia, and is given rectal salines.

Result.—In successful cases, after a decided local reaction the growth undergoes atrophy ; swallowing is restored ; and the patient's life is prolonged in comfort.

Intubation, by means of Hill's feeding tube, may be employed, if radium fails or cannot be applied. It is preferable to gastrostomy, and the tube may be worn for months.

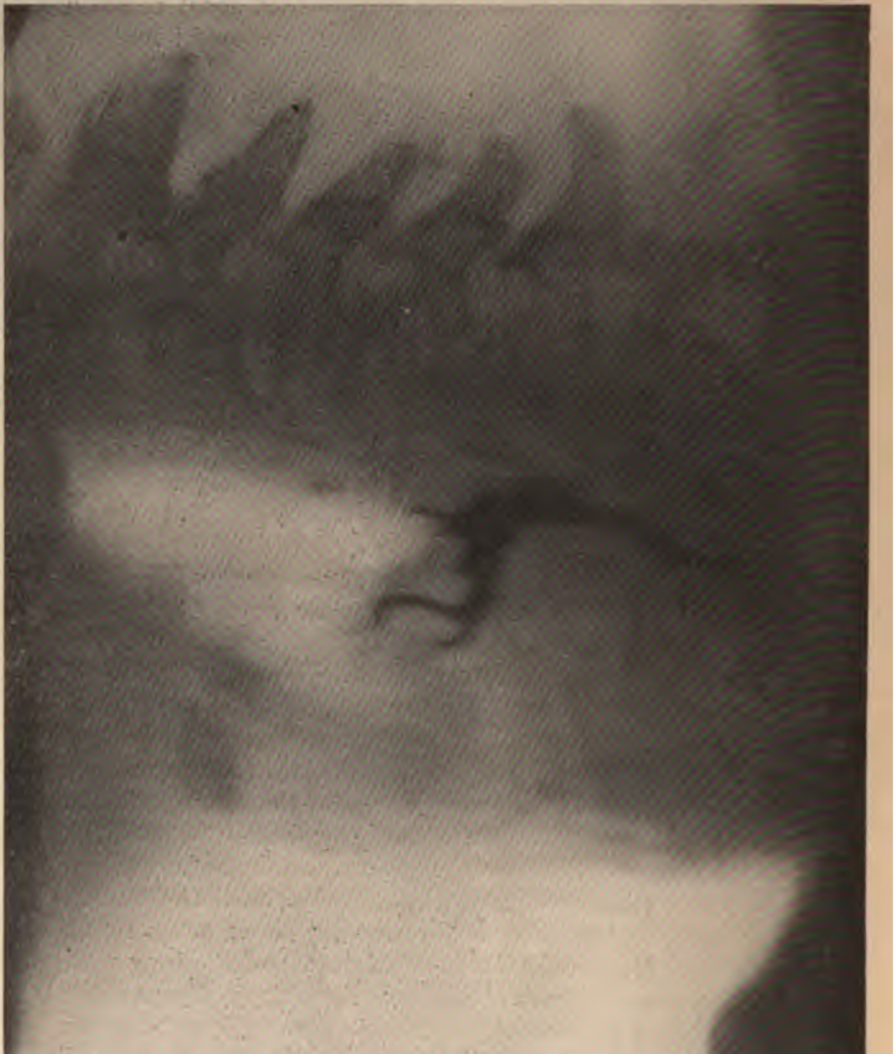
Of other palliative measures, tracheotomy may be mentioned as necessary if the growth induces respiratory obstruction.

FOREIGN BODIES IN THE ŒSOPHAGUS.

The variety of foreign bodies arrested in the œsophagus ranges from bones to coins, from artificial tooth-plates to open safety-pins. And all these can as a rule be removed by the direct method, in most cases without endangering the patient's life.

The danger of a foreign body in the œsophagus is seldom so immediate as when the air passages are the seat of lodgment, as there is rarely any risk of asphyxia from obstruction to respiration. The risk is not unknown, however, as a bulky object in the œsophagus may cause tracheal stenosis by its bulging into the tracheal lumen. But such an occurrence is exceedingly rare.

On the other hand, however, the local effects of a foreign body in the œsophagus, if it is allowed to remain there, are liable to be more immediately serious than those of a foreign body in the air-passages. The effects in both cases vary, of course, according to the kind of body present, but in the bronchi, if the foreign body does not interfere with respiration or induce acute septic infection, it will only lead to a slow ulceration of the lung with the formation of a bronchiectatic cavity,—a very disagreeable but not a dangerous malady. In the œsophagus, on the other hand, ulceration if it occurs is apt to end in rupture of the wall and fatal mediastinitis. Cases, no doubt, have been recorded of the lodgment in the gullet for many years of even such hard foreign bodies as tooth-plates, with successful removal at last by the direct method. But this kind of tolerance is very exceptional. So that although a foreign body in the œsophagus may not be so instantaneously dangerous as a foreign body in the trachea or bronchi, the period of immunity from evil consequences is not a long one, and the sooner the patient is relieved of the intruder the better.



Symptoms.—The patient is aware of a lump in the throat or chest, and he generally is quite clear that the lump felt is a foreign body which he is sure he has swallowed.

It is a rule always to accept the patient's word that a foreign body has been swallowed.

Bougies should not be used. Probangs should not be used. Coin-catchers should not be used. Because the danger to life from such instruments is greater than the danger from the foreign body.

The usual site of arrest is above or within the lower pharyngeal sphincter just at or above the upper end of the gullet, but a foreign body may be held up anywhere in the œsophagus.

Direct examination, as in other forms of œsophageal obstruction, should be preceded by an X-ray examination. (Fig. 60.)

The direct tube, as large a size as possible, having been passed down to the foreign body, the latter is seized with suitable forceps, disengaged, and removed. The manœuvre may be successfully carried out under local anæsthesia, and in simple cases takes up very little time. It is a much less delicate operation than bronchoscopy for a foreign body.

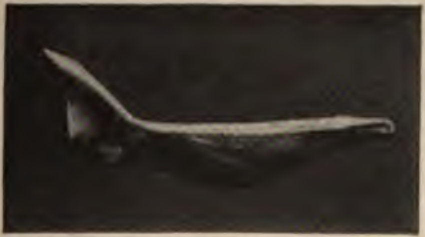


Fig. 61.—Pointed foreign body (fish-bone) successfully removed by the author from the œsophagus across the lumen of which it was lying. Natural size.

When, however, the body presents sharp pointed ends or hooks, the problem of extraction becomes difficult. From this the common sense and resourcefulness of the operator must deliver him, as rules cannot be given to apply to every kind of object.

Sharp bodies impacted across the lumen should be seized by one end, never by the middle. (See Fig. 61.)

Open safety pins, with the point upwards, should *never be pulled out.* Chevalier Jackson's method of conveying them open to the stomach, and there turning them point downward should be tried, or one of the ingenious forceps devised for closing these pins may be employed. Perhaps the most difficult objects are tooth-plates, the hooks of which have caught in the wall of the œsophagus. But these bodies may be cut in two and extracted by means of Irwin Moore's strong forceps. (Fig. 62.)

The *danger* from the presence of foreign body in the gullet, and from the manipulations needed to extract it, consists in the ease with which the soft wall of the œsophagus can be torn or penetrated.

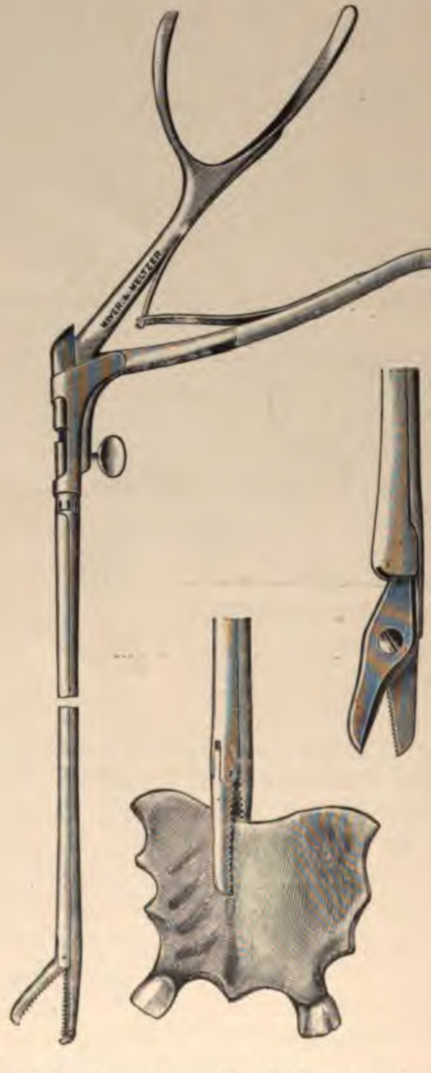


FIG. 62.—Irwin Moore's strong forceps for foreign bodies in the œsophagus; also the cutting forceps.

If rupture has taken place, nothing can be done unless the site of the rupture is in the cervical portion of the gullet, in which case an external operation should at once be undertaken, œsophagotomy performed and the wound drained. If the foreign body has not been removed, that also should be done through the external opening.

If a foreign body is situated in the cervical œsophagus, and efforts at direct extraction have failed, œsophagotomy should be performed.

In similar circumstances with the foreign body in the thoracic œsophagus, it may be possible to pass it into the stomach, where it may be left or removed by gastrotomy.

Œsophagotomy for Foreign Body.—Unless the foreign body can be felt bulging on the right side of the neck, the operation is performed on the *left* side.

The shoulders of the patient are raised, the head extended, and turned to the opposite side. An incision is made three inches long along the anterior border of the sterno-mastoid, from the level of the upper border of the thyroid cartilage downward. The skin, platysma, and superficial layer of the deep fascia having been divided, the wound is deepened along the anterior border of the sterno-mastoid until the omo-hyoid muscle crossing the wound upwards becomes visible. This muscle is cleared and drawn down or divided.

The foreign body may be palpable in the wound. The sterno-mastoid along with the carotid sheath is drawn out, and the sterno-hyoid and thyroid muscles are divided if necessary. The larynx and trachea are now tilted well over to the opposite side. Clearing the space further by blunt dissection reveals the inferior thyroid artery, and the middle and inferior thyroid veins, which should be divided between the ligatures. The œsophagus is now visible, and should be opened longitudinally on the foreign body, the incision being made as far back as possible so as to avoid the recurrent laryngeal nerve. The actual removal of the impacted foreign body calls for gentle handling, all tearing and force must be avoided.

The operation completed, the œsophageal wound is tightly sutured, and a feeding tube passed through the nose into the œsophagus past the wound and left for ten days. The wound in the neck should only be partially sutured, ample provision being made for drainage.

CHAPTER VII

EXAMINATION OF THE NOSE AND NASO-PHARYNX AND GENERAL THERAPY OF THE NOSE

First of all, inspect the outside of the nose, noting the shape of the bridge, the appearance of the skin, etc. The interior of the vestibule is examined before using the speculum by tilting upwards the tip of the nose by means of the thumb of the left hand. Note the movements of the alæ in respiration. In embarrassed breathing, especially in children, the alæ may be seen widely dilating with each inspiration. The opposite condition, a collapse of the alæ, may be observed in long-standing nasal obstruction.

Note the condition of the upper lip, excoriation and thickening of which are common in children with nasal discharges. Inspect also the eyelids, the eyes, and the eyebrows. The inner angle of the roof of the orbit should be palpated by the finger placed deeply in this angle and pressed upwards in the direction of the frontal sinus. Any swelling or fullness felt in this situation must be noted as possible evidence of frontal sinus disease.

Tapping the bone of the inner quarter of the supra-orbital ridge with the finger, as if percussing, will often elicit a complaint of pain on one or other side in cases of frontal sinusitis. The occurrence of epiphora from blocking of the nasal duct is not infrequently due to nasal disease.

The skin of the nose and vestibule may be eczematous, or the seat of acne, impetigo, herpes, lupus vulgaris, or lupus erythematodes. While the shape of the bridge varies much in different individuals, a marked irregularity in outline indicates a deviated septum or a " broken nose " from traumatism. Depression of the bridge is found in fractures and perforations of the bony septum. An abnormally broad bridge may be caused by a large middle turbinal bone, or may have resulted from distension of the middle meatus of the nose by polypoid growths.

In atrophic rhinitis a little transverse depression or dimple is frequently to be seen on the bridge about the level of the upper border of the triangular cartilage, and at the same time a certain degree of tilting upwards of the point of the nose is often present

After the external examination is completed, proceed to investigate the interior of the nose by means of a speculum. The speculum generally used at the Central London Throat and Ear Hospital is the pattern introduced by Lennox Browne, which will be found the most useful for all purposes. (Fig. 63.) Holding the speculum in the left hand, and under direct illumination from your forehead-mirror, pass the closed speculum into the nostril. Steady your left hand by resting the little finger on the bridge of the patient's nose, and gently separate the blades of the speculum. Do not introduce the blades beyond the vestibule or their opening will cause pain. Do not insert it too timidly or the vibrissæ will interfere with your view. (Sometimes it is necessary to clip these hairs.) Remember that the floor of the nasal cavity is considerably higher than the outer level of the nostril.

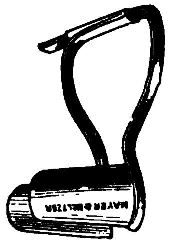


FIG. 63.--Lennox Browne's Nasal Speculum.

The first object which attracts the attention after opening the speculum is the anterior end of the *inferior turbinal body,* a red, rounded, smooth, glistening object, attached to the outer wall of the nose, and normally separated by a considerable interval from the septum, which occupies the middle line. Observe the size of this portion of the inferior turbinal, whether it fills the inferior meatus, or is in contact with the septum. Note

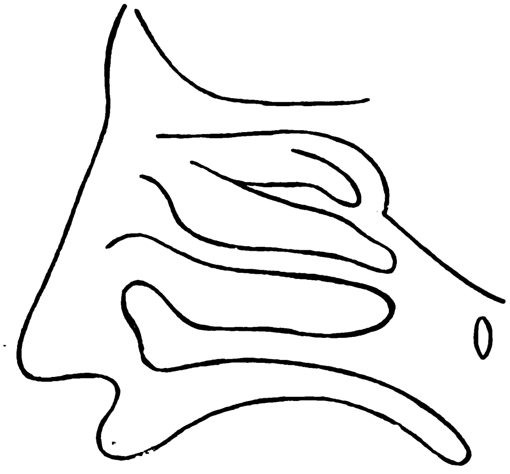


FIG. 64.—The Nasal Outline.

its colour—red or pale; and its surface—whether smooth, granular, or lobulated. By asking the patient to lower his head slightly, so as to render the floor of the nose more horizontal, you will be able, when the inferior turbinal is normal or atrophied, to follow its contour backwards towards the naso-pharynx, and, in certain cases, the red light-reflex from the posterior pharyngeal wall will be visible. Note the condition of the body, and, as far as possible, the posterior end of the turbinal. Any *synechiæ* or adhesions between the inferior turbinal and the septum, forming bridges across the inferior meatus, and often causing marked obstruction, should be noted. The examination of the posterior end of the turbinal bodies is best effected by posterior rhinoscopy, or by the naso-pharyngoscope. (See p. 219.) If the turbinal is enlarged, investigate its consistency by means of a nasal probe (see Fig. 65), most gently used, in order to decide whether the enlargement is due to osseous hypertrophy or to hyperplasia of the soft parts.

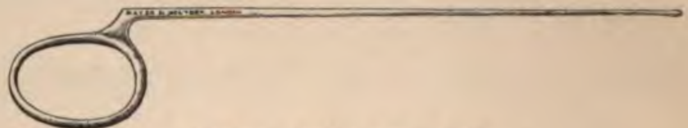


FIG. 65.—Author's ring probe.

The inferior turbinal body fulfils the function of warming, moistening, and filtering the inspired air. The anterior half is plentifully furnished with mucous glands, and these, keeping the surface constantly wet, moisten the air; while the posterior half, which is richly provided with cavernous blood-spaces, capable of rapid filling and emptying in response to changes of temperature in the air passing over them, may be regarded as a radiator supplying heat to the air. Any condition hindering or preventing the proper fulfilment of these important functions is to be regarded as disease.

Note, therefore, any *obstruction* in the inferior meatus, its size, appearance and connexions—whether turbinal, septal, polypoid, or due to a foreign body.

Note, also, the presence of pus or muco-pus in the general cavity of the nose, as it is found in acute catarrhal rhinitis. Simple chronic rhinitis does not cause a purulent discharge; if, therefore, there is a chronic muco-purulent nasal discharge, it is either due to accessory sinus suppuration or to some chronic disease, such as tuberculosis or syphilis.

" In a case of unilateral purulent discharge from the nose in a child, think of a foreign body; in an adult, think of suppuration in a sinus" (Dundas Grant).

Having investigated the inferior turbinal body and the inferior meatus, next turn your attention to the *middle turbinal*. (Fig. 14.) This structure, composed of bone covered with a much thinner layer of mucous membrane than the inferior turbinal, is the most prominent object in the nasal cavity after the inferior turbinal. Attached to the lateral wall of the nose, it occupies the greater part of the upper nasal cavity, and extends back to the posterior nares. It is normally harder and paler than the inferior turbinal, and is less influenced by vaso-motor conditions. Covered by the blunt anterior end of the middle turbinal is the *hiatus semilunaris*, a cloaca or common gutter running obliquely downwards and backwards in the lateral wall of the nose, into which open, in order from above downwards and from before backwards, the frontal nasal duct, or *infundibulum* leading to the frontal sinus and the anterior ethmoidal cells, and the *ostium maxillare* leading to the maxillary antrum. The process of bone which forms the anterior border of the hiatus semilunaris is known as the *processus unciformis*; that forming the posterior lip, and covering one of the anterior ethmoidal cells is, when enlarged, frequently known as the *bulla ethmoidalis*. The middle turbinal body is closely allied to the ethmoidal labyrinth in health and in disease, and suppuration in the osseous substance of the one is generally associated with suppuration in the other.

The presence, therefore, of pus under the middle turbinal is always indicative of sinus suppuration, either of the frontal sinus, of the anterior ethmoidal cells, or of the maxillary antrum. (See p. 326.)

The nasal chamber is divided into two parts by the middle turbinal bone—the lower, or respiratory, and the upper or olfactory. Ideally, the middle turbinal should not touch the septum anywhere, but as a matter of fact contact here and there along the free edge is the rule. Anosmia—loss or deficiency of the olfactory sense—is frequently due to abnormal enlargement of the middle turbinal.

Note, then, the size, colour, and consistency of this turbinal. Examine most carefully for purulent discharge. If pus is seen, note particularly whether it is oozing from underneath or coming over the middle turbinal from above. Note the presence of soft carious areas, bare bone, bleeding points, and polypi, which, when present, are usually rooted in this region. Do not mistake an enlarged middle turbinal for a glistening, smooth, bluish-white, movable polypus.

(There is a superior turbinal body, and sometimes yet another, higher, but in health these are not visible on anterior rhinoscopy.)

Into the spheno-ethmoidal recess posteriorly, a few millimetres from the roof, open the orifice of the posterior ethmoidal cells, directed somewhat backwards, and that of the sphenoidal sinus, opening forwards. In life the latter can only be inspected after removal of the middle turbinal. Pus from these sinuses may appear on anterior rhinoscopy as coming from above the middle turbinal, or, more usually, may be seen, on posterior rhinoscopy, or with the nasal endoscope, emerging from the upper regions of the posterior nares.

The *septum* next demands attention. Notice whether it occupies a mesial position, or whether it is convex or concave towards the side you are examining. Projections or outgrowths ("spurs") from the septum should be examined, in order to determine whether or not they act as obstacles to the passage of air through the nose. Remember that the septum consists of three main portions, osseous above and behind, where it is formed by the central plate of the ethmoid and the ala of the vomer, and cartilaginous in front, where the triangular cartilage of the nose fills in the gap between the anterior edges of the ethmoidal plate and the vomerine ala. (Fig. 75.) If the septum is deflected, note whether the deflection causes obstruction, and if so which nostril is more obstructed. Sometimes the septal mucous membrane presents a superficial excoriation or ulcer over the front of the cartilaginous portion. Note the presence of perforations of the septum, and if a perforation is present, observe its situation—whether through the cartilaginous, or, behind, through the bony septum—its size, and the condition of its edges, whether they are ulcerating or sound. Note also if there is necrosis of the bony septum. The presence of swelling in the septum—from hæmatoma following an injury, or from abscess—must be also marked.

The anterior-inferior segment of the septum, the usual site of the simple excoriation, alluded to above, is most thoroughly inspected by inserting the speculum, and then turning the patient's head around so that the profile is presented.

Next transfer the speculum to the other nostril, and examine also this side systematically. Compare the appearance of the septum on one side with that of the other, so as to be able to determine the various degrees and varieties of deviation.

In both nostrils observe the general conditions of the nasal mucous membrane—whether it appears hypertrophied or atrophied, moist or dry, red or pale, and note the presence, distribution and character of crusts.

Examine also the state of the floor of the nose. If the nasal cavity contains pus, observe very carefully from which region it seems to proceed.

Nasal Endoscopy.—By means of the straight, fine nasal endoscope (Fig. 66), recommended by the author, it is possible to examine minutely nasal regions which lie out of the range of vision on anterior or posterior rhinoscopy.

Method.—The interior of the nose is lightly anæsthetized with cocaine-adrenalin spray, and after warming, oiling and polishing the endoscope, it is passed along the floor of the nose with its window directed upwards. The inferior surface of the middle turbinal comes into view, with the space between it and the outer wall of the nose, where pus from the anterior sinus group (see p. 322) emerges, and where budding polypi make an early appearance.

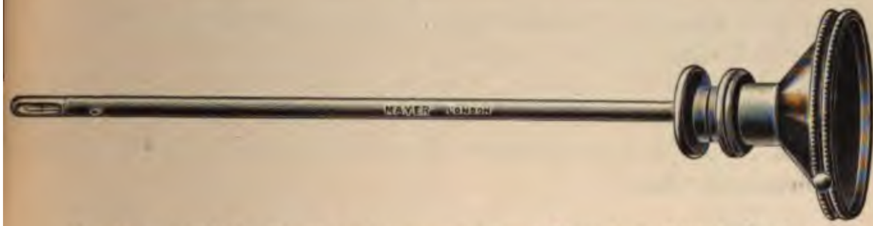


FIG. 66.—Straight endoscope for nose and naso-pharynx used by the author.

Passing backward, the spheno-ethmoidal cleft comes into the field, and here pus may be seen in cases of posterior ethmoidal or sphenoidal sinus suppuration.

The roof of the posterior choana now appears, and from it may be traced downwards the posterior edge of the septum. At this place, by turning the window towards the opposite side, the posterior end of the inferior turbinal of that side, if it is enlarged, is visible as a grey, semi-translucent granular object, which the student is liable to mistake for a polypus. (See Plate II, Fig. 4.)

Enlargement of the inferior turbinal of the side in which the endoscope is situated is seldom distinguishable, as the swollen mucosa coming into contact with the window of the instrument, altogether obscures the field.

The endoscope is particularly of value when sphenoidal sinus disease is suspected; when there is a question of small polypi hidden, it may be, behind a large middle turbinal; or when the presence of a sarcomatous or other growth in the ethmoidal region is suspected.

It is also useful after the maxillary antrum operation has been performed, as it enables us to inspect the interior of that cavity.

But we must add that considerable experience with the endoscope is necessary before one can interpret correctly the pictures it presents.

The appearances visible in the naso-pharynx on endoscopy are discussed at p. 223.

Palpation of the Interior of the Nose with the finger is seldom employed in the ordinary examination, as it is a painful proceeding. But in operating with the patient under general anæsthetic, it is of great value. The little finger (gloved) is inserted into the anterior naris, the palmar aspect being turned outwards, so that the right little finger is used for the right naris and vice versa. It is thus possible in many cases to palpate the middle meatus, and in antrum operations to hook it into that cavity. In some cases I have even been able to pass it as far as the orifice of the sphenoidal sinus.

The examination of the nose having now been completed, compare the results obtained with the observations made on the state of the mouth, pharynx, larynx, and naso-pharynx, in order that a complete mental picture of any departure from the normal may be found, for all these regions are intimately connected with one another.

EXAMINATION OF THE NASO-PHARYNX AND POSTERIOR NARES

Posterior Rhinoscopy.—Although in a systematic description of the methods employed in the examination of the upper respiratory tract an account of posterior rhinoscopy is given after the others have been discussed, it is a good rule in practice to use the post-rhinoscopic mirror early in the examination of the case, before the patient has become wearied or flustered by the examination. Otherwise gagging and retching will effectually prevent any satisfactory attempt to see the naso-pharynx and posterior regions of the nose.

To make posterior rhinoscopy successful, much practice is necessary, but time and trouble expended in learning to manipulate the mirror will amply repay their expenditure in the valuable information obtainable by this method of examination.

Either a small laryngeal mirror or the special post-rhinoscopic mirrors, such as Fränkel's or Michel's, may be employed. (Fig. 67.)

The patient should sit upright, with the head erect, or held slightly backward. Direct the patient to open the mouth without protruding the tongue. Pass a tongue-depressor into the mouth to control the tongue, and ask the patient to breathe gently. Having warmed the mirror, insert it, with the reflecting surface upwards, carefully into the mouth and back into the pharynx behind the uvula, being careful not to let it come into contact with any part—tongue, uvula, or post-pharyngeal wall. Then, when it is in position and under good illumination, slowly tilt the mirror up until it is at about an angle of 45°. This will generally bring the upper part of the posterior end of the septum nasi into view. Taking this as a landmark, the other structures can be readily found.

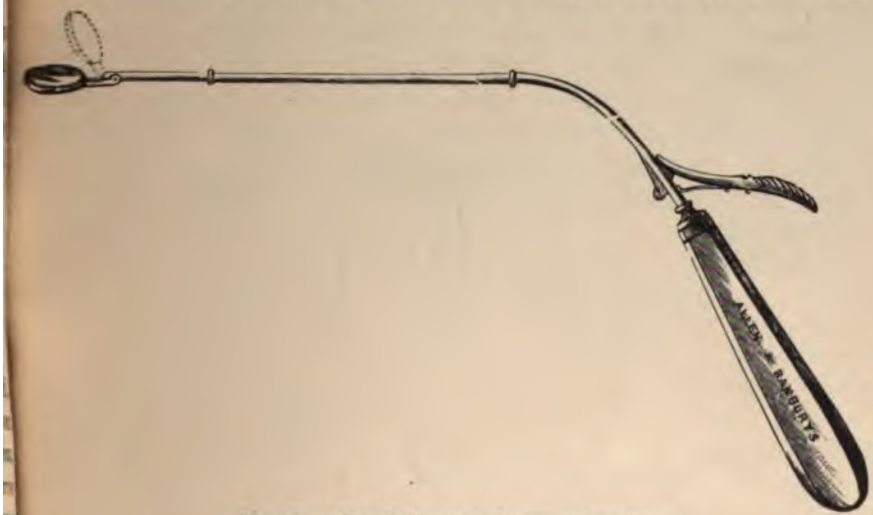


FIG. 67.—Fränkel's post-rhinoscopic mirror.

In order to view the parts, it is necessary that the soft palate be relaxed. In many cases this is not possible, and attempt at making a post-rhinoscopic examination in such patients is mere waste of time. Various instruments have been devised as palate-retractors, but, since their application as often as not leads to gagging and retching, they have not come into general use.

In an ordinary way, the following points, when attended to, will facilitate the relaxation of the palate: Firstly, make the patient breathe gently. Secondly, do not insist on the mouth being wide open. A half-open mouth will permit us to see all we want. Thirdly, do not put the tongue-depressor too far back. Fourthly, if the mouth being open leads to mouth-breathing with a raised soft-palate, ask the patient to make a " sniff " through the nose.

After the mirror has reached the pharynx behind the soft palate, it is, of course, necessary to hold it to one side of the uvula, in order to inspect the image. Withdrawal of the instrument every few seconds, in order to rest the patient, and to warm the mirror, is advisable.

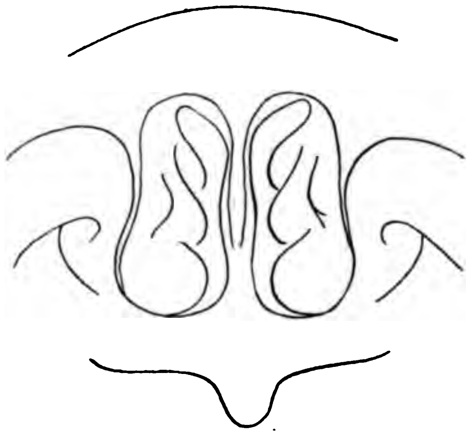


FIG. 68.—The Naso-pharyngeal Outline.

The septum nasi, then, having been found, tilt the mirror first to one side and then to the other, at the same time bringing its surface towards an angle of 90°. By this means, the posterior choana of either side will come into view, and, laterally, the posterior ends of the middle and inferior turbinals. (Fig. 68.)

The superior turbinal is occasionally seen. It is to be remembered that posteriorly the inferior turbinal does not present the bright-red appearance one marks when looking into the anterior nares, but that it is greyish-white in colour and granular on the surface. The posterior end of the inferior turbinal is frequently enlarged and sometimes pendulous, hanging down on the soft palate.

Note the presence of pus emerging from the superior or middle meatus of the nose posteriorly. Note polypi, when present, as smooth, glistening, greyish-blue bodies. Do not mistake the posterior end of the inferior turbinal, which is granular, for a polyp, which is smooth.

After the posterior nares have been inspected, tilt the mirror still more, first to one side, then to the other, in order to bring into view the orifices of the Eustachian tubes with the fossæ of Rosenmüller behind them. Sometimes in cases of middle ear suppuration the pus makes its way down the Eustachian tube, and may be seen on the orifice on posterior rhinoscopy.

Finally, let the mirror fall backwards towards the horizontal and examine the posterior pharyngeal wall as it declines. Adenoids appear as dark-red masses encroaching on the upper end of the septum. Frequently the sulci between these growths are visible, or the central recessus medius can be seen. The adenoids vary much in size; sometimes they are so large as wholly to obscure any view of the nasal septum; at other times they are only represented by a series of small furrows high up in the roof of the naso-pharynx, and between those extremes all sorts of varieties are found.

Polypoid growths or tumours springing from the vault of the pharynx may also be revealed by the posterior mirror.

Endoscopy of the Naso-Pharynx.—The endoscope having been inserted and passed through the nose to the naso-pharynx (see p. 219), the window is turned towards the opposite side in order to inspect the orifice of the Eustachian tube. (See Plate II.) In health, the orifice of the tube, with its shelving, smooth anterior lip, and its prominent curved posterior lip are clearly visible. Undue redness and swelling are to be noted. Behind the protruding Eustachian cartilage of the posterior lip is the fossa of Rosenmüller often deep and cleft-like, in which not infrequently a curled ringlet of adenoid tissue is visible (Fig. 131), even, it may be, when no adenoids can be discovered anywhere else in the naso-pharynx. Here, also, at times, cicatricial bands may be seen stretching across the fossa. These appearances may be associated with a granular or œdematous Eustachian lip, with enlargement of the posterior end of the inferior turbinal of the nose, and with deafness from catarrh of the Eustachian tube or middle ear.

Turning the eye of the endoscope round, we view now the Eustachian orifice of the same side, comparing its appearance with that of the other.

Next, the instrument is rotated so as to bring the posterior end of the septum into view about its middle, and by slewly turning the instrument, we follow the septum up to the place where it merges into the roof of the naso-pharynx.

In normal states the septum passes imperceptibly into the smooth mucous surface of the roof, but adenoids, when they are present and bulky, shut off this view, and appear as a vague, dark-red mass without definite form or limit. When adenoids are present, but scanty, they appear as small furrows, and the clefts between them are very obvious.

Rarely, the endoscope may reveal tumour formation in the naso-pharynx, but as a matter of fact, by the time the patient experiences the symptoms caused by new growth in the naso-pharynx, it has already become so large as to be invisible through the endoscope. The reason is, that when the instrument comes very close to any object the small window is occluded by the mass around it, and the whole field is obscured.

It is possible from the naso-pharynx to view the postero-superior surface of the velum palate, and also sometimes even to see as small, distant objects, the interior of the larynx and the vocal cords.

Palpation of the Naso-Pharynx is only employed when posterior rhinoscopy and endoscopy fail to afford a satisfactory view of the naso-pharynx, or when it is necessary, in order to supplement the information derived from inspection of the parts.

It is unnecessary, we may add, to palpate the naso-pharynx for adenoids in a child with enlarged tonsils, at all events, before the anæsthetic is given at operation.

Method.—The surgeon's right forefinger is guarded by a rubber finger-stall, and with a metal guard or a fold or two of bandage to protect the knuckle. The patient is seated, and his hands are held by a nurse or an assistant sitting in front of him. The surgeon stands on the patient's right, puts his left arm round the patient's neck, and brings the forefinger of his left hand into contact with the patient's left cheek. Then, on asking the patient to open his mouth, this finger pushes in the cheek between the upper and lower molar teeth, a manœuvre which keeps the mouth open during the subsequent manipulations. The guarded finger is then passed into the mouth, to the lateral wall of the pharynx, and slipped up behind the faucial pillar, and not in the middle line, as in this situation the soft palate and uvula are pushed on in front of the examining finger and block the entrance to the naso-pharynx. Having gained the naso-pharynx, the finger-point rapidly and gently feels for the septum, and then is passed into each posterior choana to ascertain the state of the turbinal bodies, and the presence of polypi, mucous or fibrous. Then the finger, turned slightly round, is moved slowly from side to side, so as to palpate the lateral and posterior walls of the pharynx. Here adenoid vegetations are felt as soft, friable bodies with vertical sulci. The presence of smooth, rounded tumours should be noted, and their attachments made out.

Take note also of the height of the pharyngeal vault, and the general roominess of the space. After withdrawal, the finger-point should be inspected for traces of blood, pus, etc.

Palpation of the naso-pharynx should never cause pain, and need not occupy more than a few seconds.

ANÆSTHETIZATION AND GENERAL THERAPY OF THE NOSE AND NASAL SINUSES

For purposes of examination, a slight spraying of the interior of the nose with 5 per cent. sol. cocaine with a little adrenalin is useful, as it causes shrivelling up of the turbinals, and permits a free inspection.

Local Operative Anæsthesia—Cocaine Tamponage.—For minor operations like the removal of polypi (see p. 275) and polypoid or papilliform enlargements of the inferior turbinal, a thin, flat piece of cotton-wool is soaked in equal quantities of sol. cocaine, 10 per cent., and sol. adrenalin, 1-1,000, and laid upon the surface to be operated upon, being spread out flat like paper on a wall. Anæsthesia, which, however, is usually but partial, is obtained in about five minutes, and lasts twenty minutes.

Cocaine-Adrenalin-Paste (O. Freer) affords by far the most complete and durable anæsthesia. The writer has been employing it for several years, and can recommend it as supplying the maximum local with the minimum constitutional effect.

It is suitable for septum operations of all kinds; for proof-puncture of the antrum; and as a local anæsthetic in sinus operations, whether a general anæsthetic is given also or not.

Used as follows, and with the patient lying down, constitutional effects are absent, notwithstanding the large quantity of pure cocaine that is used.

It is contra-indicated in polypus removal, as in that case there is usually very free mucous secretion and the cocaine paste may be displaced and swallowed.

Method.—The patient is given a preliminary hypodermic of morph. hydrochlor. gr. $\frac{1}{4}$ to $\frac{1}{3}$, with or without atropin. sulph. gr. $\frac{1}{100}$. A swab of cotton wool is twisted round a suitably long probe, and that is dipped, first of all, in pure adrenalin solution (1-1000), and applied under inspection to the mucous surface of the region of operation. From 4 to 6 grains of cocaine are placed in a porcelain receptacle, and one or two drops of the pure adrenalin solution are added to the cocaine until a paste or "mud" is formed. With this mixture, the region is painted carefully over by means of the swab, and in three minutes anæsthesia and local anæmia are produced. The anæsthesia lasts about forty minutes.

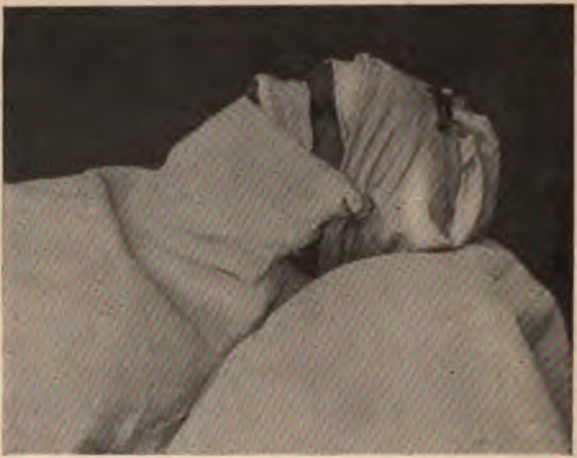


FIG. 69.—Patient Prepared for Nasal Operation.

Operations on the Nose.—It is assumed that the reader is acquainted with the aseptic technique of modern surgery. In operations on the interior of the nose, asepsis is aimed at by surrounding the nasal orifice with aseptic towels, with sterilized gauze over the mouth and bridge of the nose, and with a sterilized towel round the head. The eyes may be left uncovered. (See Fig. 69.)

Before applying the sterilized wrappings, the outside of the nose, the upper lip, chin, and particularly the skin-lined vestibule should be painted with iodine-spirit solution.

Strict asepsis should rule in the operation. The operator should wear gloves.

It is easy to infect a healthy nose with pyogenic organisms, and to substitute an ethmoidal suppuration for a deflected septum.

Nasal douching and lavage may be employed, when necessary for the removal of blood-clot or purulent discharges. The syringe recommended is Mayer and Phelps' nasal syringe (Fig. 70.) It should be boiled before use. (See p. 262.)

Packing the Nose for hæmorrhage is to be avoided whenever possible. It is painful to insert, painful to remove, and apt to infect the nose. Further, it may be followed by serious symptoms if employed when there is sinus suppuration. Nevertheless, it also is sometimes unavoidable.

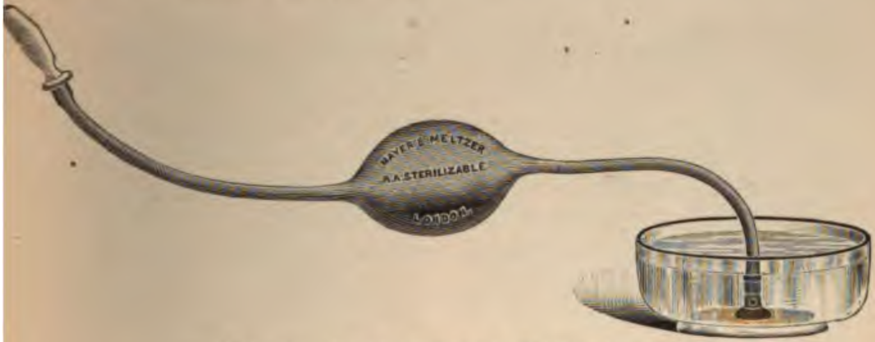


FIG. 70.—Nasal Syringe for Nasal Lavage.

Long strips of sterilized gauze are employed. The long-bladed nasal speculum is inserted, opened, and the gauze packed in by means of nasal forceps. If the bleeding point is known, local packing is usually sufficient to control it. Only in the operation of submucous resection of the nasal septum is packing of the whole cavity on both sides necessary, and this should be done around the special tubes devised by the writer. (See Fig. 85.)

Packing should not be retained for any longer than twenty-four hours. It should then be removed, a fresh pack being re-inserted, if necessary. The gauze is easier to insert and to remove if it is wrung out of sterilized liquid paraffin before use.

CHAPTER VIII

AFFECTIONS OF THE NOSE

EXTERNAL DEFECTS AND DEFORMITIES

I. **Congenital.**—Allusion is made in the section dealing with cleft-palate to the attachment of the premaxillary bone to the tip of the nose (p. 595).

In addition to that, the nose sometimes shows undue broadening with the semblance of a cleft in the middle line, as a congenital deformity, and dermoid cysts and fistulæ have been described as occurring in the middle line of the nose. Do not mistake a meningocele at the root of the nose for a dermoid cyst.

II. **Developmental.**—A not infrequent developmental deformity, and one which gives rise to nasal obstruction, is an unduly **broad columella,** whereby the anterior naris of either side may be reduced to a mere slit, the airway of which is often in these conditions further encroached upon by a prominent inner process of the lateral cartilage.

The lateral cartilage of the nose, it may be remembered, arches over the anterior naris, and by its inner process (crus mediale) helps to form the medial (internal) wall of the skin-lined vestibule. If the curve of this process is too pronounced, its free end will project the skin into the vestibule, and reduce the capacity of the air channel.

The condition is obvious on simple inspection, but if examination is limited to inspection through a speculum, it may escape notice.

The *Treatment* is operative. Under infiltration anæsthesia of the columella and vestibule with eucaine (4 per cent.) or under general anæsthesia, the patient's head being tilted well back, the skin of the unduly broad columella is closely applied to and folded tightly round the anterior end of the septal cartilage of the nose by the surgeon's finger and thumb. This will indicate how much of the redundant skin it is necessary to remove. Having been relaxed the columella is now transfixed from side to side at its upper end with a fine knife, its cutting edge downwards. With a careful sawing movement, the knife is carried down to the level of the floor of the vestibule, and withdrawn. (See Fig. 71.) The posterior lip of the incision so made is dissected back, first on one side, then on the other, until what is seen to be sufficient skin is undercut and freed. This generally resolves itself into a crescentic flap. As much of this flap as is required is cut away and removed, the raw surface left being covered by suturing the edge of the first incision at the margin of the columella, with the edge of the second incision at the margin of the mucous covering of the septum on either side. (See Fig. 72.) If the process of the lateral cartilage is projecting, it is easily exposed in the course of the dissection, and as much of it as is necessary removed by the knife or cutting forceps (Stuart Low).

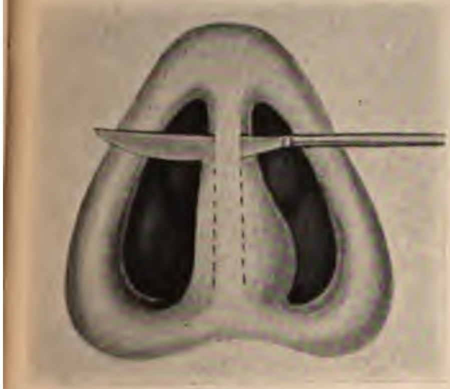


FIG. 71.—Incision for Repair of Deformity of the Columella.
(The deformity is usually bilateral. It is figured here unilateral for diagrammatic reasons).

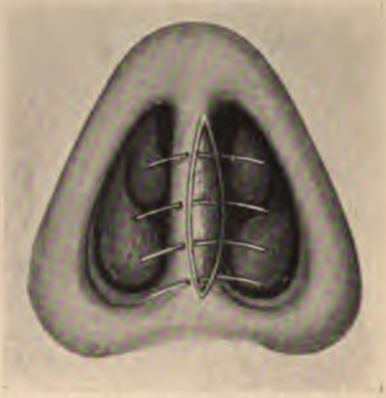


FIG. 72.—Operation for Deformity of the Columella.

High Bridge.—An unduly high or prominent nasal bridge is unsightly, and the rhinologist may be consulted with a view to its improvement. It may be remedied by

Operation.—(a) An incision is made in the middle line of the nose, the skin and soft parts are dissected off the bone, and as much of the latter is sawn or gouged off as may be necessary to remove the deformity.

This method leaves an external scar, for which reason the following operation *through* an incision in *the vestibule* is generally preferred.

The first steps of this method constitute the *normal for all operations* undertaken with the object of *remedying bridge deformities*. Consequently they will now be described once and for all.

(b) A general anæsthetic is necessary. The nasal vestibule is clipped of its vibrissæ and carefully painted with iodine. With a sharp knife an incision is made (see Fig. 73) through the roof and outer wall of the skin-lined vestibule of one side, the incision being deepened under the guidance of the finger externally until the point of the knife reaches the outer subcutaneous surface of the nasal bone of the same side. A curved periosteal elevator is now introduced, and the soft parts over the entire bridge of the nose elevated subcutaneously from the nasal bones, and, if necessary from the ascending process of the superior maxilla. (See Fig. 74.)

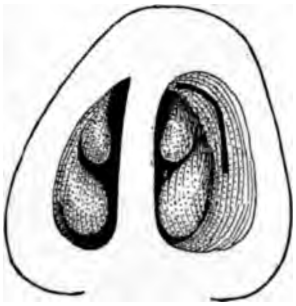


FIG. 73.—Normal Incision in the Roof and Outer Wall of the Skin-lined Vestibule for access to the bridge of the nose in the subcutaneous operations for bridge deformities.

This gives free access to the bridge, and the subsequent steps depend upon the type of deformity.

When the bridge is unduly high, a fine saw, or gouge, or Ballenger's reverse chisel is employed to reduce it to the desired height.

After the deformity has been reduced or repaired, the patient's head is turned on one side, and the wound freely flushed with normal saline solution to wash out debris and blood clot.

Finally, the bridge of the nose is covered with a collodion dressing, while a firm roll of lint is placed against either side of the nose and secured with strips of plaster, so as to keep up pressure and lessen the tendency to hæmatoma and sepsis.

A dry packing in the vestibule is sufficient to seal the vestibular incision.

Difficulties and Dangers.—The separation of the soft parts is easy, but considerable experience is necessary to estimate how much or how little of the bridge is to be removed (or, it may be, to be supplied).

The chief danger is hæmatoma after operation, and subsequent ptic infection of the extravasated blood, and as the face is exceedingly vascular, this accident is very prone to occur and may spoil the best-planned operation, especially when a cartilage or bone-graft has been inserted, as in the treatment of the deformities about to be detailed.

It is best prevented by careful dressing, firmly applied as described above.

Results.—In normal conditions, the results are very satisfactory.

III. Acquired Deformities and War Injuries.

The "Pinched" or Narrow Nose is a common consequence of neglected adenoids in childhood and the consequent mouth-breathing, forming one of the features of what has been termed by the writer "the rodent face." Owing to the constantly open mouth, and the consequent withdrawal from the upper jaw of the expansile moulding action of the tongue, coupled with the incessant drag and pressure of the muscles and soft parts of the cheek on the developing alveolar processes, a lofty or "Gothic" palatal arch is produced (Fig. 129), in consequence of which the permanent teeth of the upper jaw, and especially the incisors, erupt irregularly and are cramped and crowded together, projecting below and beyond the upper lip, and failing to meet the lower incisors in biting ("the open bite"). The whole superior maxilla being narrowed, the nose is pinched, the nasal cavities are laterally

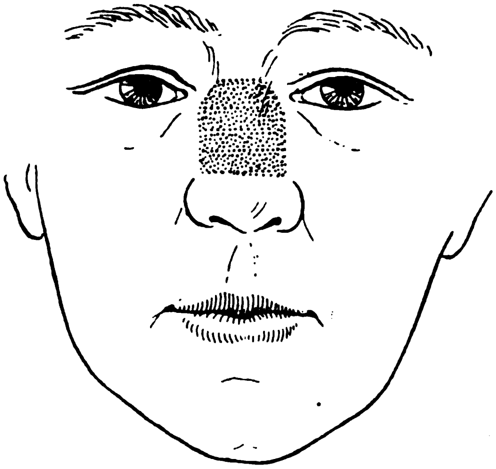


FIG. 74.—The shaded area indicates the extent of the bridge of the nose bared subcutaneously through the vestibular incision (Fig. 74) in operations to remedy bridge deformities.

compressed, and their breathing capacity is very seriously reduced. The limited nasal capacity is moreover still further diminished by deviation of the septum, which is almost invariable in these cases, while to complete the picture, it is quite common to find, even in adults, the peccant adenoids still occupying the naso-pharynx.

Symptoms.—The result of this chain of malformations is, in addition to the unsightliness of "the rodent face," the persistence into adult life of mouth-breathing, with all its attendant discomforts and dangers.

Treatment.—Apart from prevention by early removal of adenoids, treatment of the deformity should be initiated as soon as it is observed.

The aid of the dentist should be sought to rectify the irregularity of erupting or erupted teeth. In established cases, the deformity is irremediable as such, but efforts should even then be made by the dentist to secure the ability to close the mouth easily, and by the rhinologist to render the nasal cavities as roomy as possible.

Adenoids (and tonsils, if present) are removed; the columella, if thickened, is reduced as described at pp. 228-29; and the septum is straightened by submucous resection. In extreme cases, where such measures have failed to confer the ability to breathe through the nose, I have, once or twice, made a large perforation in the septum from near the anterior to the posterior naris, in order to give more nasal capacity. But the benefit was trifling, though the result proved the experiment to have been harmless. Rhinitis sicca did not ensue.

Depressions of the Bridge of the Nose.—The group of deformities comprised under this heading are such as are caused by trauma, as (1) of a blow fracturing and flattening the nasal bones and fracturing or bending the septum; (2) of a hæmatoma abscess of the septum whereby a large septal fenestra is produced owing to necrosis of the septal cartilage (see p. 250); or (3) by the destructive action of tertiary syphilis on the skeleton of the nose (see p. 289) (saddle-bag nose, etc.).

These different conditions are grouped together here because the deformities they produce are of the same type in that the bridge of the nose is depressed or flattened and the treatment consists in building it up again.

Treatment—Paraffin Injections.—The injection of solid paraffin by means of a special syringe into the loose tissues of the bridge is alone worthy of consideration, as the injection of liquefied paraffin brings with it the danger of embolism. And even the solid paraffin is nowadays seldom employed, as experience has shown that in course of time it evinces a disposition to spread about the face, reproducing the old, and introducing some new deformities, besides acting as a tissue-irritant. (Wyatt Wingrave.)

Cartilage or Bone Grafting has taken the place of the paraffin injection. Cartilage is perhaps preferable to bone, as the latter is said to undergo slow absorption in the tissues. The cartilage may be conveniently supplied from the nasal septum, if, as is frequently the case, the septum is being operated on by submucous resection at the same time. Otherwise, a suitable strip may be obtained, in young people, from a costal cartilage. The strip of cartilage should be oblong, and flat or rounded, being pared as near to the size desired as possible. Edges and corners should be rounded off.

The first step of undermining the skin and subcutaneous tissue of the nasal bridge through the vestibular incision should be completed before the cartilage is cut, so as to obtain the latter fresh. If removed first, it must be carefully preserved in warm, normal saline. The cartilage graft is inserted through the vestibular incision, and manipulated into place by the fingers externally. The compresses and dressings are applied as already described.

The *result* is often surprisingly good, but considerable experience in these operations is necessary.

Lateral Displacement of the External Nose, such as is produced by a blow or a fall on one side of the face and bridge, may be considerably improved by passing a fine saw through the vestibular incision, after the soft tissues have been undermined, and sawing through, on the convex side, the nasal bone and nasal process of the superior maxilla—avoiding injury to orbit and lachrymal apparatus—and subsequently pressing the nose over to that side.

Defects of the External Nose from War Injuries.—Loss of substance from the nose may extend up to complete loss of the feature. Treatment is reserved for cases where the deformity is grievous, as where there is a defect in the nose leading direct to the interior, and where portions of the nose, or the whole organ, are lost. The detailed treatment lying beyond the scope of this book, we shall limit ourselves to general principles.

Defects of the bridge opening directly into the nasal chamber may be closed, as Andrew Wylie was the first to point out, by loosening the middle turbinal partly from its attachment, and " advancing " it to fill the gap. Thereafter, a flap of skin may be cut from the cheek or forehead, turned round and sutured to the margins of the opening.

Loss of the entire organ with the laying open of the nasal chamber has been remedied by the brilliant work of Major Gillies and his colleagues. By " advancing " the turbinals, and by cutting strips and flaps of septal cartilage, together with flaps cut from the forehead, or cut from and held in place by the forearm of the patient, they have been able to restore the nasal prominence and organ to such a degree as to rid these patients of the haunting sense of a repulsive deformity.

Readers interested in this side of the work are referred to the Proceedings of Roy. Soc. Med. Laryng. Sect., 1917.

Web of the Vestibule.—Sometimes the vestibule of one or both nostrils is partially occluded by a web-like band of integument springing from the floor of the nostril. It may be removed with the knife, the raw surface left being sutured or covered with a skin-graft.

DEFORMITIES AND DEFECTS OF THE INTERIOR OF THE NOSE

Cellular Distension of the Middle Turbinal.—The middle turbinal is a process of the ethmoid, and in the development of ethmoidal cells about puberty, a cell may form in the middle turbinal and distend that structure so much as to cause a bending of the septum to the opposite side, and even a bulging of the nasal process of the superior maxilla externally. It seems to be commonest in females, and it occasionally sets up severe and continuous pain at the side or over the bridge of the nose.

On *inspection*, the cause of the pain is obvious in the smooth, hard enlargement of the anterior end of the middle turbinal.

Diagnosis.—It is distinguished from nasal polypus by being red, hard, and immovable. From mucocele of the ethmoidal region, and from enlargement of the bulla ethmoidalis, it may not be distinguishable until after operation.

Treatment.—Anæsthetize by cocaine-adrenalin paste (see

p. 225); or general anæsthesia by nitrous oxide may be employed.

Luc's or the writer's nasal forceps (Fig. 94) are introduced into the nose, and made to take a full grasp of the bulbous anterior end of the middle turbinal. The forceps are closed, quickly twisted round, and withdrawn. Two bites may be necessary if the cell is a large one.

Or, the method employed in snaring a large middle turbinal may be tried, although the bulk of the bone to be removed renders the snare unsuitable for this variety of enlargement. (See p. 339.)

In a manner analogous to the production of the foregoing deformity, the *bulla ethmoidalis* may undergo excessive development, and may even extend down into the inferior meatus. By the enlargement of the bulla, the middle turbinal is compressed against the septum and out of sight, and it is only after removal of the distended cellular bone that a proper diagnosis can be made, for the hypertrophied bulla is almost invariably mistaken for the middle turbinal.

It may be removed under cocaine by the writer's forceps.

Occlusion of the Posterior Choana by a diaphragm, partly membranous, partly bony, sometimes is met with, usually on one side only.

Symptoms.—The patient is totally unable to breathe through the affected nostril, or to blow it, and in consequence, it is constantly filled with a glairy mucus like starch, which flows out of the nose when the head is held forward.

The diagnosis is easily made on posterior rhinoscopy, the closed choana being seen in the mirror. It often shows a central dimple or depression. It may be distinguished from cicatricial closure by the membranous feel of the occluding diaphragm, on palpation of the naso-pharynx.

Treatment.—Under general or local anæsthesia, the diaphragm is incised as fully as possible with a sharp stout scalpel, and then by means of a long burr, its bony edges are rubbed down level with the walls of the nose. There is a tendency for the membrane to form again, but this can be avoided by removing a piece of the posterior end of the nasal septum with a pair of Löwenberg or Jurasz forceps introduced through the mouth and naso-pharynx.

DEVIATIONS AND SPURS OF THE NASAL SEPTUM

In the adult nose a certain degree of septal deviation is normal. Abnormality is reached when the deviation is so considerable as to cause nasal obstruction or to excite reflex neuroses. The same rule holds good regarding spurs and outgrowths from the septum.

Etiology.—Deviations of the septum are developmental or traumatic.

Developmental Deviation is seldom seen before the age of seven years. It is predisposed to by the presence of adenoids, and occurs in the typical " rodent face " deformity. But it is also common in people who have never suffered from any other effects of adenoids, and, as it is more frequently met with in civilized than in savage races, we may suppose that its frequent occurrence in the former is due, in some way, to their larger cranial development.

The varieties of deviated septum and septal spur are many. It suffices, as a rule, to note to which side the septum is deviated, together with the degree of occlusion of nostril present. Deflections are usually most marked in the triangular cartilage, which on section appears as a simple arc, with the concavity towards one nostril and the convexity towards the other. It occasionally happens that the anterior end of this arc is formed by the extreme anterior end of the septal cartilage, the so-called " columnar " cartilage, and when this occurs, it involves a displacement of the columnar cartilage from the columella. This deformity, sometimes known as *displacement of the columnar cartilage,* is visible from without on ordinary inspection of the nose, and may produce obstruction in the vestibule. This type requires a special modification of the submucous resection operation for its removal. (See later, p. 248.)

A combination of deviation of the septal cartilage (and it may be also of the vomerine septum) with a *spur* is often met with, in what is known as the *oblique ascending spur,* an outgrowth of cartilage (the cartilage of the vomer) along the lower edge of the septal cartilage, that passes upwards and backwards between the central plate of the ethmoid and the vomer. (See Fig. 75.) This spur begins close to the anterior end of the septum, and near to the floor of the nose, and it is a useful landmark in operating (albeit sometimes difficult to negotiate) as it marks the upper edge of the vomer ; that is to say, the line where the cartilage meets the bone of the vomerine septum.

The oblique ascending spur varies greatly in prominence. Sometimes it is no more than a mere boss or moulding on the septum ; at other times it projects right across the nasal passage as a sharp spine, which may even be closely in contact with the outer wall of the nose. It is characteristic also of this spur for it to become more prominent as it passes backward, so that we not infrequently find the nasal passage narrowed to the degree of obliteration about the position of the posterior end of the middle turbinal. Such obstruction may altogether escape notice on casual examination.

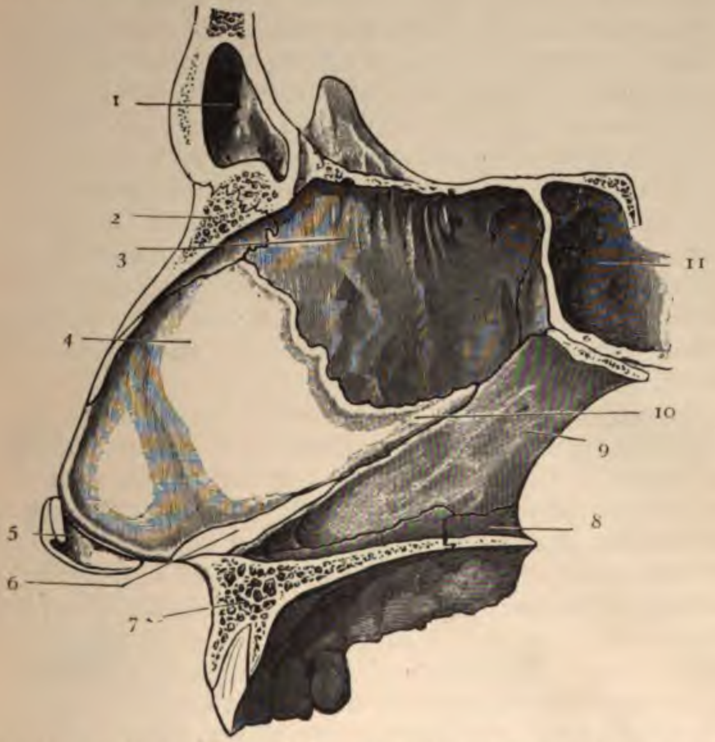


FIG. 75.—The Nasal Septum. (From Cunningham's Text-book of Anatomy.)

(1) Frontal sinus. (2) Nasal bone. (3) Central plate of ethmoid. (4) Quadrilateral cartilage of the septum. (5) Lateral cartilage. (6) Vomerine cartilage. (7) Superior maxilla. (8) Palate bone. (9) Vomer. (10) Cartilage of the vomer (or sphenoidal process). (11) Sphenoidal sinus.

Another spur of importance, both as a cause of obstruction and as requiring special attention in the septum operation, is what is known as the *basal spur*, which is a unilateral hypertrophy or exaggeration of the mesial palatal ridge of the superior maxilla, most marked towards the front of the septum. When it is present, the septal cartilage is often bent in such a way that the lower border of the cartilage is displaced towards the opposite side of the nose from the basal spur, and thus there is formed a deep recess, fossa, or gutter in the septum, in the depths of which the muco-perichondrium is adherent, and is thus liable to be torn through in the operation. The basal spur is bony.

The foregoing varieties of deviation are developmental in their origin and nature, but in addition to these we encounter bends, sometimes very extreme and irregular, which are the effect of *traumatism*: the so-called *traumatic deviation*.

For convenience we shall deal with it also at this place.

In the commonest type of **traumatic deviation,** the bend is vertical, that is to say, it runs up and down; and it is limited to the anterior portion of the septal cartilage. Often this deflection is so extreme as to block the nostril entirely, and the bend is then usually quite angular—obviously the result of a blow on the point or to one side of the nose, "buckling" or fracturing the cartilage. And yet posterior to the deflection the septum may be quite straight. Occasionally, however, we come across cases in which this type of traumatic deviation is combined with the developmental variety, and with an oblique ascending spur.

As might be expected, traumatism is responsible for the irregularly contorted septa that come for treatment, and as these are generally the after-effects of quite serious injuries, the structure of the septum is frequently very irregular. At bends and angles, areas of adhesion between the cartilage and its perichondrium, the results of fractures or of hæmatomata, are encountered in operating, and add to the difficulties and tedium of our task.

Symptoms.—The effect of deviation is to narrow one or both sides of the nose, and the narrowing may be so considerable as seriously to reduce the capacity of the airway. When the obstruction is unilateral, it might be supposed that the increased width of the opposite side would compensate for the narrowing. And this may be true to begin with as regards the septum, but under these conditions, and probably in response to the stimulus of the increased quantity of air that is passing in and out of the wider nostril, the turbinals of that side undergo hypertrophy, and in course of time come actually to block this, the originally open side, and so it often happens that the side the patient feels obstructed is not the side of the septal convexity, but that of the septal concavity.

Thus a deviated septum in causing *unilateral* obstruction leads to hypertrophic rhinitis in the wider side, and so, it may be in course of time, to all the phenomena and sequelæ of chronic *bilateral* nasal obstruction.

Another common effect of deflection of the septum, with or without spur formation, is interference with the drainage, and, perhaps, also with the ventilation of the nasal accessory sinuses. This is evident from the fact that a grossly deflected septum is frequently found in association with acute sinus suppuration on the narrowed side. Obviously, the encroachment upon the free space of the middle meatus of the nose by the displaced septum limits the room available for swelling of the mucosa about the ethmoidal region, and so favours the occurrence of acute sinusitis. To a similar effect, also, we may attribute the occurrence of polypoid disease in cases with septal deviation. For it is noteworthy that the polypi, as a rule, make their first appearance upon the narrow side of the nose.

A third group of symptoms is at times attributable to septal deflection, especially when the septum or a part of it, such as a prominent sharp spur, is in actual contact with a turbinal or with some part of the outer wall of the nose. This group is that of reflex irritation. Thus we find reflected pain and headache (L. H. Pegler): irritable cough; sneezing; paroxysmal rhinorrhœa, and even, at times, such remote reflexes as asthma, cured by septal resection and the separation of points that have been in contact.

We must add a note of caution at this juncture. When these phenomena are present, the septum operation is justifiable, indeed it is necessary. But the surgeon should be careful not to promise too much. Sometimes the operation is successfully accomplished, and yet the pain, cough, asthma, or what not, continue in spite of all.

Treatment.—We operate to straighten a deflected septum when,

(a) It is causing nasal obstruction on one or both sides.

(b) It is associated with polypus formation or sinus disease.

(c) It seems to be the source of reflex irritation.

(d) It prevents access in operations on the antrum, on the frontal, ethmoidal, or sphenoidal sinuses; or on the lacrymal sac.

(e) By its effects, it seems to be producing catarrh of the Eustachian tube and middle ear, or when it prevents the passage of the Eustachian catheter, if the use of that instrument is likely to cure the deafness.

(f) In chronic laryngitis; and in some cases of papilloma of the larynx and tuberculous laryngitis when there is some nasal obstruction.

(g) In recent traumatism with hæmatoma of the septum.

Operation on the septum also forms the preliminary step to the trans-nasal operation on the pituitary body. (See p. 312.)

Contra-Indications—Local.—Operation is contra-indicated if the deviation is producing no symptoms, and in bilateral atrophic rhinitis; acute rhinitis; and syphilis of the nose or pharynx.

General.—Diabetes and asthenic conditions generally.

Nasal sinus suppuration is *not* a contra-indication.

SUBMUCOUS RESECTION OF THE NASAL SEPTUM

(See Figs. 76, 77, 78, 79, 80, 81, 82.)

Description.—The raising of the mucous membrane (muco-perichondrium and muco-periosteum) of both sides of the septum through a single unilateral incision, and the removal of as much of the contained cartilage and bone (Fig. 76) as is necessary to rectify the septal deformity and to restore free bilateral nasal breathing.

Anæsthesia. (1) *Local.*—(a) By means of cocaine-adrenalin paste as described on p. 225.

In making the application particular attention may, with advantage, be paid to the region of the naso-palatnie nerve on either side of the septum.

(b) By the application of tampons of cotton-wool soaked in 10 per cent. sol. cocaine and 1-1000 sol. adrenalin.

The former is recommended as more satisfactory and safer.

(2) *General.*—For patients who desire a general anæsthetic, chloroform is given. But in this also, the septum must be treated with cocaine-adrenalin prior to operating, in order to secure a dry, bloodless field. A useful plan is to make one application of the cocaine-adrenalin paste immediately before the general anæsthetic is started.

In children, cocaine should be avoided. Adrenalin only should be applied, in the 1-1000 strength a few minutes before operating, as its effects are evanescent in children.

Position.—The patient lies on the operating table on his back, with the head slightly raised. The surgeon stands on the right-hand side. Rigid aseptic technique should be adhered to throughout. The illumination must be as bright as can be got.

Operation.—The nasal speculum is inserted into the nostril of the *convex* side of the deviation, and a long gauze strip is gently packed into the posterior region of the nose, in order to prevent blood finding its way back into the throat.

With a short-bladed knife, a curved incision is made through the mucous membrane and perichondrium of the septum down to the cartilage. The incision is carried from near the roof of the vestibule down to the floor of the nose, and lies just behind the skin-mucous membrane junction. (Fig. 77.)

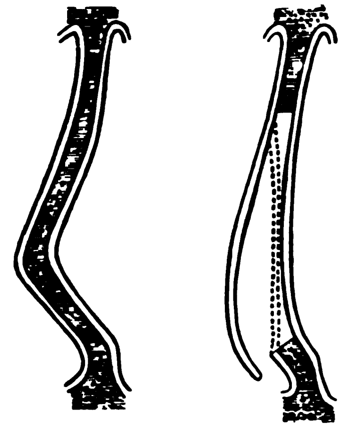


FIG. 76.—Submucous Resection of the Nasal Septum. Removal of cartilage. (Diagrammatic—after Laurens.)

(When there is a prominent, sharp, bony spur present, it is often advisable to reverse the direction of the incision, beginning below on the floor of the nose and carrying it up to near the roof of the vestibule.)

This incision, through which all subsequent manipulation is to be carried out, should be deepened to the surface of the cartilage.

A suitable elevator (Greville Macdonald's recommended) is now passed under the posterior lip of the incision, and the muco-perichondrium is raised from the cartilage. (Fig. 78.)

Normally, at the site of the incision adhesions to the cartilage render this separation difficult for a moment or two, but by keeping assiduously close to the cartilage, a line is reached where the separation can be effected with ease, the elevator passing backward, upward and downward almost of its own weight.

No force should be used, and if an obstacle is encountered it is left for the time being, note being made of its situation for later treatment. The usual position for such an obstacle is at the junction of the septal cartilage with the palatal ridge of the superior maxilla (the basal spur). To this we shall recur.

As much muco-perichondrium is raised as will give free access to the deviated part of the septum. (See Fig. 83,) That done, a blunt-pointed knife is taken, and the cartilage itself is incised from above downwards in the line of the original mucous membrane incision. This carries us through the cartilage to its opposite side, and in making this cut, care must be taken not to wound the mucosa of that side.

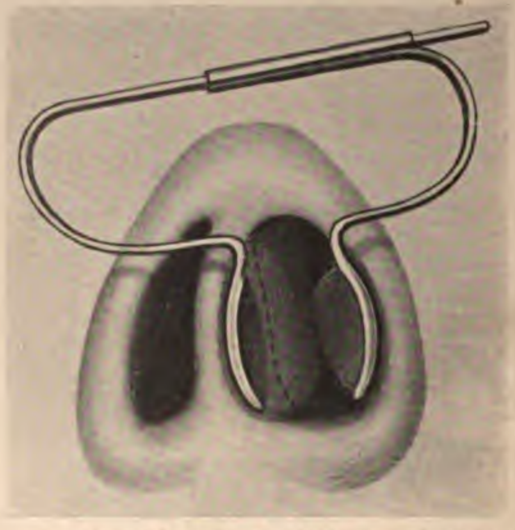


FIG. 77.—Submucous Resection of the Nasal Septum. (1) The line of incision.

By the pressure of a finger in the opposite nostril, the cartilage is now pushed over into the original wound, so as to make its cut edge present, and under this cut edge is slipped again the elevator in order to elevate the muco-perichondrium of the opposite side. (Fig. 79.) This elevation is most safely and readily accomplished under inspection, the speculum being changed to the corresponding side of the nose. The area of muco-perichondrium raised corresponds to that of the first side.

A long-bladed speculum (see Fig. 80) is now slipped between the lips of the incision and passed in between the cartilage and the loosened muco-perichondrium, one blade being on one side, and the other on the other, so that when the blades of the speculum are opened, the cartilage is seen isolated between them. (See Fig. 80.)

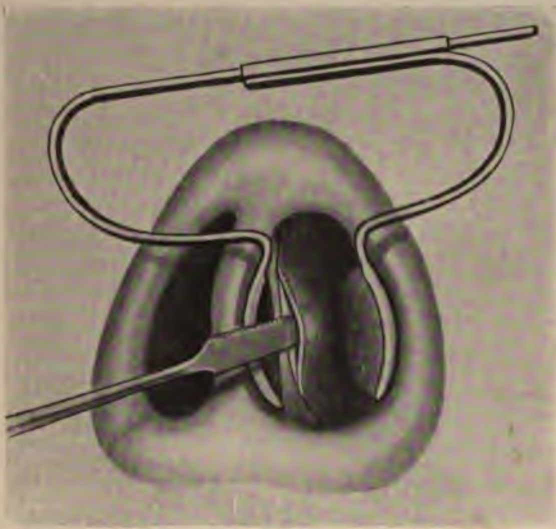


FIG. 78.—Submucous Resection of the Nasal Septum. (2) Introduction of the elevator.

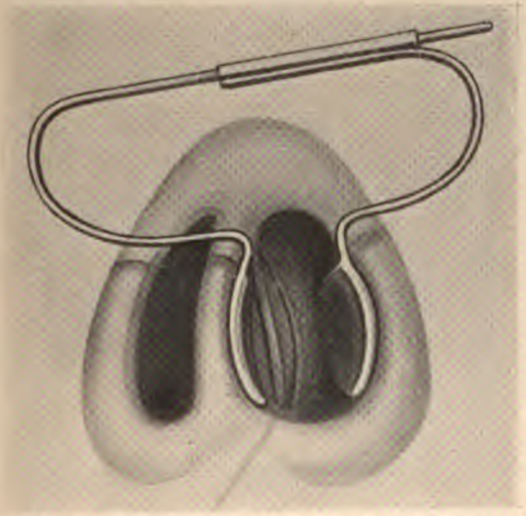


FIG. 79.—Submucous Resection of the Nasal Septum. (3) The mucosa on the convex side has been separated; the cartilage has been cut through and its cut edge pushed over to make it present in the wound.

With cutting forceps or with Ballenger's swivel-knife as much as is desired of the cartilage and bone thus isolated is removed. This usually carries us up to the level of the middle turbinal above, and to the anterior edge of the vomer behind, taking also, if convenient, the oblique ascending strip of cartilage. (Figs. 75 and 83.)

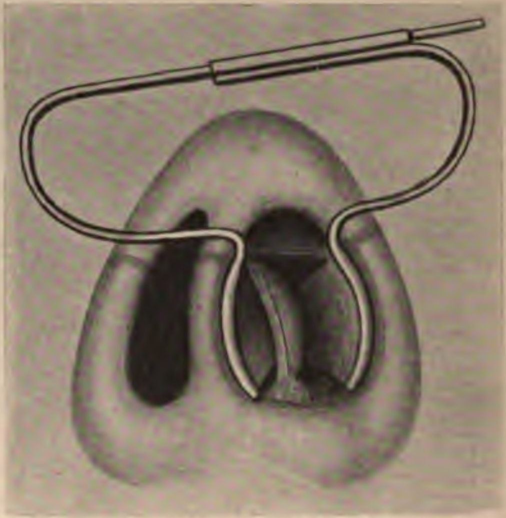


FIG. 80.—Submucous Resection of the Nasal Septum. (4) The long-bladed speculum has been inserted and opened so as to raise the loosened mucosa of both sides from off the septal cartilage, thus isolating the cartilage.

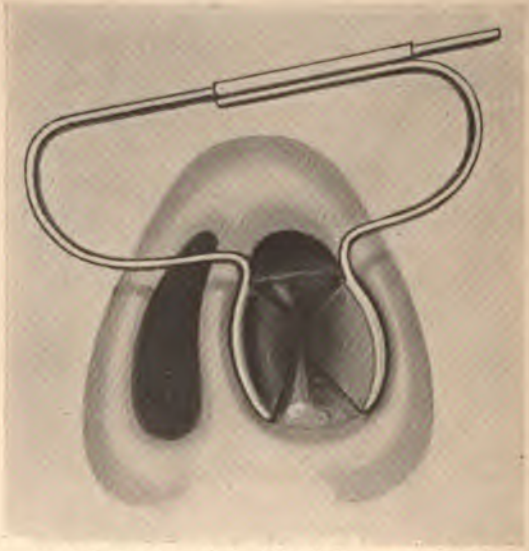


FIG. 81.—Submucous Resection of the Nasal Septum. (5) View after removal of the isolated septal cartilage.

We now return to the *adhesion* along the line of the *basal spur*.

Anatomically, the periosteum of the basal spur, which, as we have seen, is composed of bone, passes over the spur from one side to the other without a break, and thus the sub-perichondrial space of the septal cartilage is not continuous with the subperiosteal space over the bone of the spur, and in elevating the coverings of the septum, before we can pass from the cartilage to the bone of the basal spur, this layer of tough periosteum has to be traversed. The speediest way of doing so is to cut through it with a sharp knife, cutting on to the bone, and in such a direction as not to button-hole the mucous membrane. If this is postponed until part of the cartilage has been removed, it may be done under inspection. Sometimes, however, it is more convenient to make the division at an earlier stage, and even, it may be, at the very start of the operation.

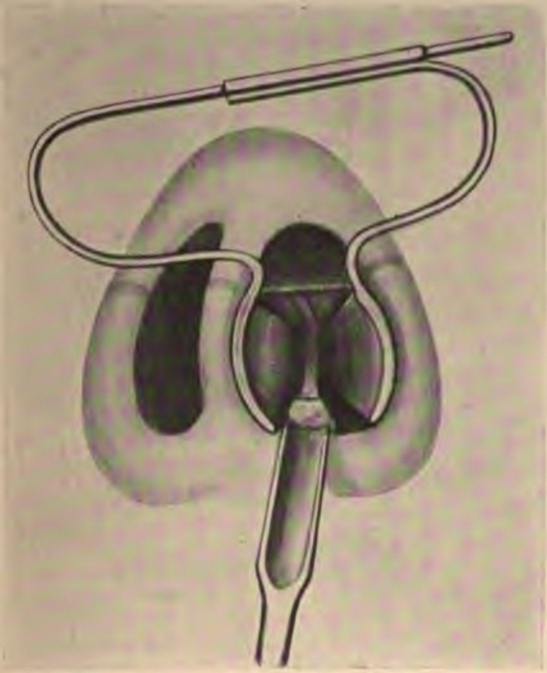


FIG. 82.—Submucous Resection of the Nasal Septum. (6) The gouge applied for the removal of the basal spur of bone.

Once this periosteum is incised, the elevator, inserted once more, raises it from the basal spur, first from one side, then from the other, and after it is bared, with a bent square-ended gouge (Fig. 84), the bony spur is undercut from before backwards; levered up; and removed. (Fig. 82.)

The long speculum is now withdrawn from the nose, the flaps are allowed to fall together, and by means of the ordinary short speculum, the nasal cavity is inspected, in order to see whether enough has been removed to provide a free airway on either side. As a rule, two further portions of the septum require attention, (1) posteriorly in the vomerine region, especially on the middle turbinal level, where the remains of an oblique ascending spur still need attention; and (2) anteriorly, at the upper end of the original incision, where some of the edge of the cartilage still needs trimming.

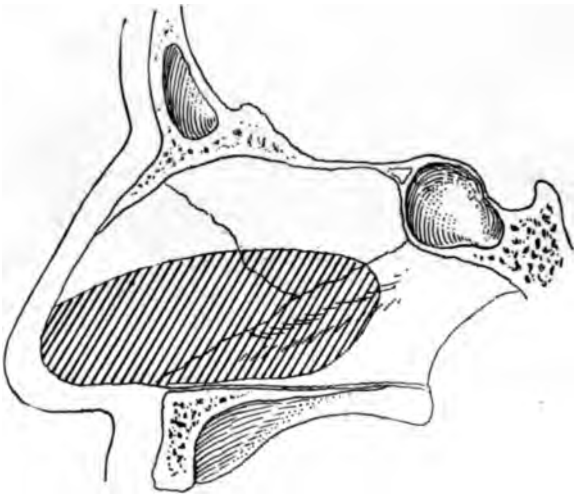


FIG. 83.—Diagram showing the extent of the cartilage and bone usually removed in submucous resection of the nasal septum. (In operating on the pituitary body the removal is carried back as far as the sphenoidal sinus—see p. 312.)

The long-bladed speculum is therefore re-inserted, and those parts dealt with by means of cutting forceps.

Finally, the septal wound-cavity is freed of chips of bone and cartilage, and of blood-clot, the flaps are allowed to fall together, and a packing of strips of sterilized gauze, wrung out of liquid paraffin, is inserted into the nasal chamber on either side to prevent the formation of a hæmatoma between the flaps.

The packing remains *in situ* for twenty-four hours: it is then removed, and not replaced unless there is hæmorrhage. The packing is very irksome to the patient. For this reason the writer places metal tubes (Fig. 85) in the nose for breathing purposes, and inserts the packing around them. The tubes are kept clear of blood and discharge by being swabbed frequently with hydrogen peroxide solution.

After-treatment is simple. When the packing is removed, the nose is douched with normal saline solution, and thereafter it is left alone.

After the packing has been removed, a reactionary swelling of the turbinals comes on, and leads to blocking of both sides; this continues during the healing of the wound for about a week or ten days, so that the patient does not begin to appreciate the benefits of the operation for at least a fortnight.

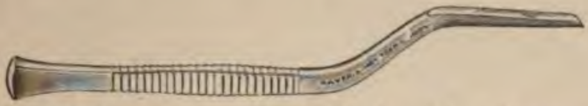


FIG. 84.—Killian's bayonet gouge (for the basal spur in submucous resection).

The wound usually heals without any trouble, but convalescence may be interrupted by a rise of temperature usually from septic tonsillitis. (See p. 28.) This is due to septic infection from the nasal wound, and can be avoided by attention to asepsis before and during operation. It is best treated by antistreptococcus serum (20 c.cs.) hypodermically.

Difficulties.—The operation presents many pitfalls and difficulties, which the operator must learn in time satisfactorily to circumvent.

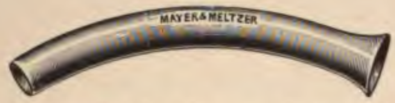


FIG. 85.—Author's metal tube, for use after submucous resection.

Button-holing the flap is the most frequent accident, but it is negligible unless the button-hole extends during the operation, and becomes a large gap. Sometimes, as when there is a sharp-edged or sharp-pointed spur, button-holing can scarcely be avoided, but in such cases, one may prevent its occurrence by postponing the elevation of the muco-perichondrium along the summit of the spur, until the cartilage around it has been removed, or the opposite plan may be adopted of raising the periosteum over the basal spur of the bone before undermining the septal perichondrium.

If a button-hole in one flap corresponds in position to a button-hole in the other, a perforation in the septum will be left after the wound is healed. The operation being planned to prevent such an eventuality, its occurrence, artistically speaking, is annoying, but, fortunately, such a perforation often gives excellent breathing-space, although it may also favour crust-formation. (See p. 281.)

The formation of synechiæ or adhesions is prevented by avoiding button-holing and by light-handed manipulation, so as to avoid ruffling the mucosa of the outer wall of the nose.

Modifications.—No two nasal septa are alike, and each requires some special modification of the general plan.

Lateral Displacement of the Columnar Cartilage with Deviation of the Septum.—In this variety the anterior end of the septal cartilage is apparent, projecting into the vestibule of one nostril, while the convexity of the deflected septum lies as a rule in the opposite side of the nose. For this type, two incisions are recommended. *A*, Down the anterior edge of the displaced cartilage in the vestibule, and *B*, on the opposite side further back on the convexity of the septum inside the nose. (See Fig. 86.) Through *A* as much of the displaced cartilage as projects into the vestibule is removed, *and no more*. Incision *B* gives access to the septum inside the nose, and through it the rest of the operation proceeds as in ordinary cases, only cartilage lying posterior to incision *B* being removed. Thus, there is left in the middle line, between incision *B* and the wound *A* in the opposite vestibule, a strip or pillar of cartilage and this

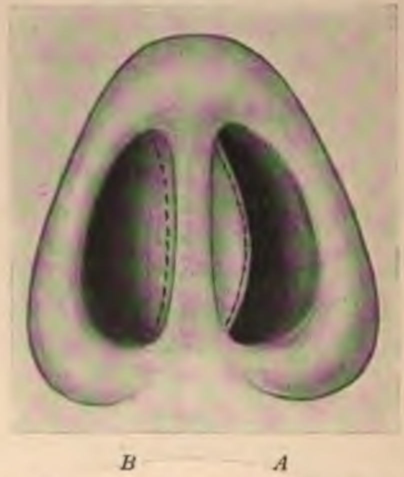


FIG. 86.—Submucous Resection of the Nasal Septum. (7) The double incision for cases with displacement of the cartilage at the columella.

serves to support the tip of the nose, which, if the operation were carried entirely from incision *A* right backwards, might show some flattening.

In General,—when deflection angles are acute, when there are, as in traumatic cases, unexpected adhesions between perichondrium and cartilage, and in all difficult cases, the rule is, to be content with limited separations of perichondrium and piece-meal removals of cartilage and bone, proceeding step by step, and declining swift or spectacular progress.

Cautions.—Avoid removing the cartilage close under the bridge. Otherwise, sinking of the bridge may ensue and some external deformity.

Avoid operating in active syphilitic conditions, as destructive ulceration and deformity may follow for which the operator will be blamed.

The more perfect the adrenalin-hæmostasis, the easier is the operation.

It is generally agreed that the operation should not be performed on children under twelve unless unavoidable, as the growth of the nose may be stunted.

If one inferior turbinal has undergone hypertrophy, anterior turbinotomy may be required to restore nasal breathing in addition to the septum operation.

Submucous Resection of Spurs.—Sometimes an outstanding spur may be removed without resecting the septum itself. A vertical incision is made in front of the spur, the mucosa of the septum covering it is elevated, and the underlying prominent cartilage and bone is removed by knife, forceps, or saw.

The old method of sawing off these spurs as a whole is not recommended, as it may cause a large perforation, or leave an extensive scar to favour crust-formation.

The *Results* of submucous resection are pleasing, especially as regards nasal obstruction and its effects.

When the operation is performed for old-established deafness, however, as from chronic catarrh of the middle ear, striking benefit can scarcely be expected.

The establishment of free nasal respiration has a beneficial effect upon such laryngeal diseases as chronic laryngitis, pachydermia, papilloma, and even tuberculosis of the larynx.

PERFORATIONS OF THE NASAL SEPTUM

are not uncommon, and may arise either from injury or from destructive disease of the septum.

In **Simple Perforation** which affects the cartilaginous septum only, especially about Little's area (see p. 255), we have to deal with a perforation due, it is generally supposed, to simple ulceration from crust-formation and picking with the finger-nail. The long continuance of trivial injuries to the mucous covering of the septum produced by this habit initiates and keeps up a simple ulcerative process, which in time eats its way through the septum. (Fig. 87.)

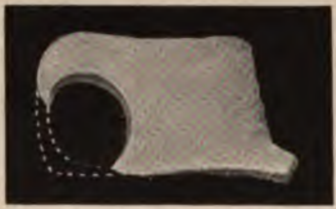


FIG. 87.—Cartilage of the Nasal Septum removed by submucous resection, showing fenestra. (For the specimen of which this a drawnig I am indebted to Mr. C. Horsford.)

The writer suggests that, before the septum perforates, its cartilage becomes fenestrated from pressure-atrophy induced by the dried mucous crusts adhering to the mucosa. (See also p. 280.) Nose-picking will induce ulceration and also epistaxis, but perforation is not produced, he believes, unless the cartilage has previously undergone fenestration.

The pre-eminence of dust deposit in the etiology of the simple perforation is obvious, from the frequent occurrence of the defect in stone-masons, and other workers in dusty surroundings, and if the dust be of a chemically irritating or destructive material, such as chronic acid, or common salt (D. M.) its ulcerative and perforating properties are all the greater.

Septal perforation is also apt to follow the injudicious application of the galvano-cautery or powerful caustics like silver nitrate to the cartilaginous septum.

It is characteristic of perforation from such causes (with the exception of the chronic acid type) that they affect the cartilaginous septum only. The stronger and more vascular osseous septum is able to resist them.

External injury, such as a blow on the nose, producing hæmatoma abscess of the septum (see p. 253), in consequence of which the cartilage of the septum undergoes dissolution, may lead to perforation, but as a matter of fact, perforation from this cause is relatively rare, although extensive cartilage destruction is common enough, since the muco-perichondrium remains intact—upon one side at least.

We have already seen that the submucous resection of the septum may cause perforation, but it is much less liable to happen than it used to be when spurs were sawn off entire.

Among the local destructive *diseases* that produce perforation of the septum, the commonest is syphilis, which may obliterate the whole of the structure, both bony and cartilaginous. It is, indeed, the distinguishing point between a simple and a syphilitic perforation that the former affects the cartilage of the septum only, while the latter attacks bone and cartilage, either indifferently or with a preference for bone.

Tuberculosis, lupus, and cancer deposits on the septum lead to its perforation, but care should be exercised not to mistake a perforation due to those diseases for a perforation upon the edges of which those diseases are deposited. If a growth or nodule affects a segment only of the rim of a perforation which is otherwise cicatrized, then the perforation is the primary and the neoplasm or granuloma the secondary condition.

Finally, in gravely debilitating diseases, such as typhoid fever and diabetes, more or less of the cartilaginous septum may slough, and, if the patient recovers, a perforation will be left.

Symptoms.—A perforation of the septum may give rise to no symptoms whatever. On the contrary, its presence may actually be an advantage in providing more nasal space for respiration.

Otherwise, the small, simple perforation sometimes makes its presence known by producing a whistling noise during inspiration, a symptom often very irritating to the patient. Larger perforations encourage crust deposit upon their edges, in consequence of which epistaxis may result. Finally, the extensive perforations of syphilis, by withdrawing from the bridge the support of the septum, are liable to be followed by falling-in of the bridge and deformity. Exactly how much of the septum need be lost to produce this deformity does not seem to be quite clear, and as great defects may exist without deformity, there is perhaps no general rule governing its occurrence. But when the bony septum is involved, and when the cartilaginous septum close under the bridge is destroyed, deformity may be expected. The fact that it often delays its appearance until after convalescence in syphilitic cases suggests that the falling-in may be due chiefly to cicatricial contraction.

Treatment.—The cause of the perforation must, of course, be removed, if that is not already in abeyance.

The perforation as such need not be interfered with if it is causing no symptoms. A whistling perforation may be cured by a plastic operation, or by enlarging the perforation, if the former is out of the question.

The **Plastic Closure** of small perforations is effected as follows:

The edge of the perforation is rawed. A flap of mucosa is cut on one side, as in Fig. 89 *A*, slid *forward* over the perforation, and sutured with fine catgut into position. On the opposite side, the flap is cut as in Fig. 88 *B*, slid *backward* over the perforation and sutured.

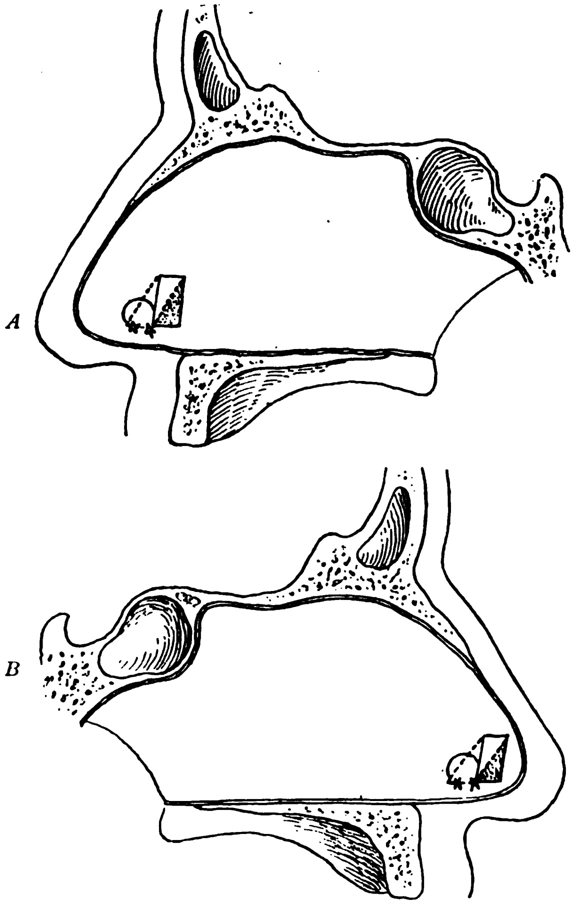


FIG. 88.—Closure of a small simple perforation of the septum by alternate flaps.

Large perforations cannot be closed, but their edges should be kept moistened with liquid paraffin or some simple ointment.

INJURIES OF THE NOSE AND EPISTAXIS

Fracture of the Nasal Bones is always the result of blows on the bridge of the nose, generally near the root.

The *Symptoms* are pain, swelling and deformity, and perhaps crepitation.

Immediately after the blow is received, the deformity is visible, but it is soon masked by the swelling, which forms with very great rapidity, and it does not become obvious again until the swelling has passed away, by which time the fracture has united and the deformity is fixed.

The type of deformity depends upon the direction of the blow. If it falls directly on the bridge, the nasal bones are broken more or less transversely, and the bridge is flattened and widened. If it falls to one side or the other, the nasal bones are broken obliquely, and the bridge presents lateral displacement, usually with the *convexity on the same side that the blow was received*, and the flattening on the opposite side.

With the fracture of the nasal bones and their displacement, there is always associated a bending or a fracture of the septum, either of the cartilaginous or of the osseous portion or of both, and the septal lesion is frequently associated with the formation of septal hæmatoma, which, like the external swelling, conceals for a time the presence of the septal deformity.

Fracture of the perpendicular plate of the ethmoid may be an accident of the utmost gravity if the force has been sufficient to cause fracture also of the cribriform plate, and the opening of the anterior cranial fossa. But the force necessary to inflict injury so deep must be very great.

Hæmatoma and Abscess of the Septum.—Hæmatoma of the septum results from a blow on the nose. It shows as a dark red swelling of the septum, frequently bilateral.

Search in these cases should be made for signs of fracture of the septum and of the nasal bones. If left *in situ* the effused blood is slowly absorbed, and then the fracture, if present, may for the first time become evident in the deformity of the septum.

Occasionally it happens that these blood swellings are transformed into abscess from infection through the intact mucous membrane. The swelling then increases, becoming tense, bright red, very tender, and it is associated with constitutional disturbance. Liquefaction of the cartilage with the formation of a large fenestration may ensue, as a result of which the deformity of the nose is likely to be aggravated.

Treatment of Fracture of the Nose.—Far too little attention is paid to this injury.

1. If seen immediately after the injury is received, while the deformity is still visible, the patient should be anæsthetized, and efforts made to rectify, first of all, the external deformity by external manipulation aided if necessary by padded elevators and similar flat instruments passed inside the nose. The septum should also be straightened if possible. It is generally easy to retain the replaced fragments in their proper position, and the attempt should always be made, sterilized gauze plugs being inserted into the nose, as is done after submucous resection, to aid in keeping the fragments of the nasal bones in position, and also to prevent the formation of hæmatoma of the septum. A dressing should be applied externally of the same kind as is used in the operation on deformities of the bridge. (See p. 230.)

The internal packing may be retained for several days as its presence is not intolerable, but hæmatoma of the septum is less prone to develop after forty-eight hours.

2. When the external swelling has appeared before the case is seen, X-ray examination may give some information as to the site and nature of the fracture and displacement, but skiagrams of the region in question are not easy to interpret.

It is possible, however, to mould the fragments from without into good position as late as the tenth day after injury.

3. If the septum is occupied by a hæmatoma, an incision should be made under local anæsthesia through the mucosa as if for the submucous resection, and the blood and blood-clot entirely evacuated. The long-bladed speculum is inserted, or the finger may be passed into the cavity, and the state of the septal cartilage and bone investigated. If they are found to be fractured and displaced, attempts should be made to readjust the deformity, the nose being finally packed as after submucous resection.

4. If hæmatoma-abscess has formed by the time the patient is first seen, it should at once be opened, and the contents evacuated, fragments of cartilage and bone that may be present being removed, as they will no longer be viable. Packing is not employed, as the cavity must be allowed to drain.

Such deformities as are then present must be dealt with after recovery, as outlined on p. 233.

Epistaxis, or nose-bleeding, is caused by (1) injuries from blows, etc.; (2) local affections, such as ulcers, simple or malignant, membranous rhinitis, foreign bodies, etc.; (3) systematic affections, typhoid and other fevers, anæmia, hæmophilia, purpura, and diseases characterized by high arterial tension such as Bright's disease, atheroma, hepatic cirrhosis, etc.

The usual site of bleeding is on the anterior inferior angle of the triangular cartilage, a spot where the bleeding point is easily seen. At this spot, which is known as *Little's area*, a small anastomotic vessel runs which is liable to be wounded in the detaching by the finger-nail of the fine mucous crusts that tend to adhere about this part; or the vessel may be broached by a shallow simple ulceration that readily forms here; or it may rupture and the bleeding break out spontaneously.

The amount of blood that can be lost from such a minute vessel is surprising, and when actually confronted with a lively epistaxis, the patient bleeding freely and apparently from both nostrils, it seems absurd to suppose that only this small vessel should be the sole source of the flow. Nevertheless, in 99 per cent. of the cases of epistaxis this is the site of bleeding, and here it may at once be stopped.

When due to a constitutional disorder, the hæmorrhage sometimes springs from several points at once. Frequently recurring attacks of epistaxis, a local cause being absent, should lead to an examination of the patient for signs of Bright's disease or hepatic cirrhosis. In such cases, epistaxis is often credited with being a safety valve, relieving high tension in the circulatory system, and perhaps in this way postponing or preventing the rupture of an artery in the brain. But this safety-valve idea is overdone, and patients are too often permitted to become seriously anæmic through repeated epistaxis before any attempt is made to bring the hæmorrhage to a standstill.

It should be remembered that recurrent epistaxis is also one of the usual signs of malignant disease in the nose. (See p. 292.)

Treatment.—Find the bleeding spot, and seal the vessel.

In most cases this can be done by pressure applied upon Little's area, and even pressing in the flaccid ala nasi upon the septum may be sufficient. The patient can do it for himself.

When the bleeding is in progress, examine the nose with the aid of mirror and speculum, mopping the blood dry with sterile gauze.

If the bleeding spot is situated in Little's area, apply to it a tampon of cotton-wool, soaked in sol. adrenalin (1–1000), and held in apposition with a firm packing of gauze in the vestibule and floor of the nose.

Remove this in five minutes, and the bleeding will be found to have stopped, so that you can inspect the vessel. Dry the surface by mopping with gauze, and apply along the course of the vessel by means of a cotton-tipped probe, 50 per cent. sol. argent. nitrat. in aq. The solid crystal may be used upon the osseous part of the septum, but it is too strong for the cartilaginous septum, and may induce its perforation.

The galvano-cautery point, at a dull-red heat, may be employed instead of the chemical caustic, but it is more difficult to control, and it is apt to start the bleeding again.

The nose should never be plugged as a whole for bleeding from Little's area.

When hæmorrhage seems to be springing from the upper regions of the nose, and the bleeding-point cannot be seen, these upper regions can be securely packed off with ribbon-gauze inserted under inspection, and the inferior meatus left free of packing.

Hæmorrhage from operation wounds is dealt with on the same principle; the packing is applied to the region affected.

The control of epistaxis by a plug in the posterior and one in the anterior naris, the nasal cavity and its sinuses being allowed to fill with blood, is a blind and blundering method of treatment

The posterior naris may be plugged for bleeding after removal of the posterior end of the inferior turbinal, but when it is, no other plug should be needed.

DISEASES OF THE VESTIBULE OF THE NOSE

The vestibule being lined with skin, it is liable to skin disease.

Eczema of the vestibule is caused by irritating nasal discharges —as, in a mild and transitory form, from acute coryza, and, more severe, in nasal diphtheria. It is also seen sometimes in paroxysmal rhinorrhœa and other conditions that produce a free flow of liquid from the nose, and the writer has reported its occurrence as a result of spraying the nose with strong solutions of menthol.

The *symptoms* are those of eczema elsewhere with a special tendency to the formation of painful fissures, for which reason care must be taken not to open the blades of the examining speculum as wide as usual.

Treatment.—The cause must be removed, after which the skin of the vestibule may be treated with

℞. Ichthyol. 10 per cent.
P. Zinc. Oxid., gr. 20
Vaselin. ad ℥i.

If there is much infiltration and fissures are deep and sore, the vestibule should be first of all painted with sol. argent. nit. (gr. xx. to ℥i.).

Small fissures in and about the vestibule, particularly in the recess under the tip of the nose, form a favourite portal for the introduction of the *erysipelatous infection*, and people subject to recurrences of facial erysipelas will be found nearly always to owe their attacks to a breach of the surface in this situation. Here also, in many cases, is to be found the first deposit of the lupus nodule in lupus attacking the skin of the face. (See p. 290.)

Syphilis affects the vestibule in two forms. First of all, the primary chancre, from finger infection, may appear in the vestibule as a chronic sluggish infiltration coming, if untreated, to produce great swelling and deformity of the parts. The slow progress, the obstinacy to simple remedies, and the enlargement of submaxillary lymphatic glands should lead to diagnosis. (See also p. 287.)

As a late secondary or tertiary lesion, symmetrical *ulceration of the vestibule* both of the interior and of the borders of the alæ and adjoining surface of the upper lip occasionally appears. It readily yields to treatment if recognized in time, but may be mistaken for simple eczema, and lead to loss of tissue and to deformity, before the error is rectified.

Furunculosis of the vestibule is not uncommon, the boil being situated in a hair follicle. It gives rise to heat, pain and swelling, and runs the usual course of furunculosis with the tendency to indefinite recurrence either locally or elsewhere.

If seen in an early stage, the boil may be aborted by carefully puncturing the centre with a hypodermic needle, and injecting a minute quantity of pure phenol. The needle should not penetrate beyond the zone of subcutaneous infiltration. It is a painful treatment, but effective.

Once the boil is formed, incision and curetting under an anæsthetic is recommended.

Prevention is best secured by the local daily application of Ungt. hydrarg. oxid. flav.

In obstinate cases, a stock staphylococcus vaccine may be tried.

Rhinophyma is the name given to the unsightly nodulo-bulbous hypertrophy of the skin of the exterior of the nose, which is due to an adenomatous overgrowth of the sebaceous glands, and is met with as a sequela to the severer forms of acne.

It is best treated by operation. Under a general anæsthetic, the redundant skin and tissue is freely shaved off, sufficient being removed to reduce the organ to something like its pristine dimensions. Hæmorrhage is not excessive, and the raw surface readily heals without skin grafts, although many surgeons do make use of them.

DISEASES OF THE INTERIOR OF THE NOSE

ACUTE CATARRHAL RHINITIS (INCLUDING EPIDEMIC CATARRH; ACUTE CORYZA; "COLD IN THE HEAD"; ACUTE PURULENT RHINITIS)

Epidemic Catarrh.—The importance of epidemic catarrh as the starting point of many serious diseases of the nose, throat and ear can scarcely be over-estimated. Hitherto, far too little attention has been paid by epidemiologists to this widespread endemic disease, the sequelæ of which entail much ill-health, suffering, and disablement.

Etiology.—The disease is an infectious malady, travelling from one person to another by contact. The bacterial cause is probably unknown, as from four to six micro-organisms are variously blamed for its occurrence. Clinical and personal experience, however, proves beyond doubt that the distribution and the occurrence of the disease is occasioned, in present conditions, less by an omnipresent organism than by the varying degree of resistance in the individual. Some people enjoy a life-long immunity from the disease; others never seem to be free of it. But the majority of people are attacked about twice a year—roughly, every six months. That is to say, the immunity conferred by one attack is exhausted in about six months' time.

It seems also to be the case, however, that a permanent immunity is ultimately acquired by those who are susceptible to the infection, as one frequently learns from patients that as years go on, the attacks become less frequent and less severe until they finally disappear. But it unfortunately is often the case that by the time the immunity is acquired, the patient has already become the victim of the serious secondary effects of the disease, such as deafness, nasal sinus suppuration and polypus formation, or chronic bronchitis and asthma.

The old belief that epidemic catarrh is due to exposure to cold is untenable in the face of the facts that it is found in all climates, and at all seasons of the year indifferently. The feeling of shivering in the early stages is an effect, not the cause of the disease. While it is true, moreover, that prior to the days of modern rhinology, many conditions were believed to be "cold" which we now diagnose as specific nasal and naso-pharyngeal diseases, still it is probably also the case that sufferers from such chronic diseases as sinus suppuration and adenoids are more susceptible to the infection, and more frequently attacked by it than are individuals whose upper respiratory passages are healthy. Such patients frequently fall victims, either because the local resistance in the nose is reduced by the chronic disease, or because the recurring attacks of "catarrh" are due, not to the epidemic virus, but to a temporary rise in virulence of septic organisms already resident in the nasal sinuses, or in the adenoids.

Chronic rhinitis from whatever cause arising—deviations of the septum; polypi; sinus suppuration—frequently becomes acute from slight causes, and in this condition it probably is true that a chill from exposure or getting wet is sufficient to precipitate active symptoms, sometimes even with pyrexia.

In children, frequent "colds" are almost invariably the result of adenoids, the chronic sepsis of which becomes acute on slight provocation, or without any provocation at all.

Apart from the virus of epidemic catarrh, and the effect of such diseases as those we have just mentioned, symptoms of acute rhinitis occur also as prodromal or early phenomena of the *exanthemata*, especially of measles and whooping-cough. True influenza also may produce acute nasal catarrh, but the ordinary epidemic nasal catarrh is undoubtedly a disease quite distinct from influenza.

A severe gonococcal rhinitis in new-born infants is described.

Finally, acute rhinitis may be induced by exposure to irritating gases, vapours and dust; and by taking iodides internally.

Thus, the group of symptoms known as "acute catarrhal rhinitis" or "acute coryza" may be due to a large number of different causes.

Pathology.—Catarrh of the mucous-lined cavities of the upper respiratory tract is a surface infection, and the inflammatory reaction extends to no great depth; at all events, in simple and early cases. The reaction produces congestion and swelling of the mucosa with a surface exudate of thin, watery secretion mingled with pus cells. The secretion of the mucous glands is diminished in the early and increased in the later stages.

Symptoms—Epidemic Catarrh.—The disease begins in one circumscribed area, in the naso-pharynx or nose or larynx, and spreads from it. Victims of the disease know by experience when an attack is beginning, the affected locality, which is at first quite small in extent, being the seat of a peculiarly characteristic irritation.

When the larynx is selected, the symptoms develop into acute catarrhal laryngitis (see p. 97), but the nose, in such cases, also becomes affected.

When it starts in the nose or naso-pharynx, it sets up, during the first twenty-four or forty-eight hours, nasal stuffiness from turbinal swelling; with acrid nasal watery secretion; fullness in the head and ears; and general malaise, the last varying in severity with the extent and intensity of the local phenomena. Any remedy which relieves the local conditions benefits the general. But the constitutional disturbance is more subjective than objective, for although the skin is hot and dry, and the pulse a little increased, yet the temperature is seldom raised in uncomplicated cases, and appetite for food is unchanged or increased.

The signs of inflammation tend to involve the nose, larynx, trachea, and bronchi, and may last from two to six weeks in a susceptible subject or in a neglected case.

According to the classical description of acute catarrh, the first period of scanty secretion is followed by a period of profuse watery discharge, and this by a period of purulent discharge, but in reality such clear distinction is seldom attainable, for the discharge varies in character and in quantity from hour to hour.

The period of recovery, which is liable to interruption by relapses, is marked, however, by the purulent nasal secretion becoming more copious and more mucous in character, and finally by its drying up. During all the stages, the loss of vaso-motor control of the turbinals from the action of the bacterial toxines upsets the balance of turbinal function, so that at one moment the nose is unduly patent and streaming with watery secretion, and at the next it is absolutely blocked and dry. The latter condition is most noticeable and persistent when the patient is recumbent, and it interferes both with rest and sleep.

The complications of acute epidemic catarrh include laryngitis (see p. 97), tracheitis and bronchitis; acute suppuration of the nasal accessory sinuses (see p. 316); acute catarrh and suppuration of the middle ear; while among its sequelæ we find chronic and hypertrophic rhinitis, and chronic exudative catarrh of the middle ear, with all the consequences of these conditions.

The symptoms of the other varieties of catarrh are less severe and persistent than those we have just been describing—that is to say if the cause is removed.

Diagnosis.—True epidemic catarrh should not be confused with the acute exacerbation of nasal sinus suppuration or with adenoiditis in children.

There is no such disease as a "chronic cold." When catarrhal symptoms are chronic, the nose or naso-pharynx is the seat of real disease, the most usual being ethmoiditis in adults, and adenoiditis in children.

Prognosis.—In aged and debilitated persons, an attack of acute catarrh is dangerous; in sufferers from chronic bronchitis and asthma, it means a serious temporary aggravation of all their symptoms, and the same applies to patients with chronic nasal sinusitis and middle ear disease. Thus, deafness is always made worse by a cold, and the loss so sustained may not be restored, while patients who have been operated on for nasal sinus suppuration frequently experience a recurrence of their old symptoms during acute coryza. Finally, the liability to reinfection during catarrhal attacks renders it important to shut off the mastoid operation cavity from the naso-pharynx by a sealing up of the Eustachian tube.

Treatment—Preventive.—The infection of epidemic catarrh is probably most lively in the early stages of the attack, and during that period the patient ought, if possible, to be isolated. At the present day such an injunction will not be followed, but the future will certainly see attempts made to control an infectious disease which at present is a cause of much unnecessary morbidity, and, indirectly, of much mortality.

The patient, however, can attend to the following rules:—

He should avoid close contact with other persons, as in kissing.

The spraying of his environment with droplets of saliva as he is speaking can be prevented by his wearing a light veil or mask of cotton over the mouth.

Nasal discharges being highly infectious, should be blown into paper or rag handkerchiefs and burned.

And cups, spoons, and other vehicles and vessels used by him should be sterilized.

Curative.—An attack can sometimes be aborted if, soon after the prodromal irritation in nose or naso-pharynx is first felt, the nose is freely syringed with warm, sterilized, normal saline solution.

And indeed, even after the disease has become fully developed, nasal syringing twice daily with warm saline, or, if the discharge is thick, with ʒiv. of

℞.	P. Potass. Chlor.,	ʒ iv.
	P. Sod. Bibor.,	ʒ iv.
	P. Potass. Bicarb.,	ʒ iv.
	P. Sacch. Alb.,	℥ i.—M.

in a pint of warm water is very soothing to the irritated and inflamed mucous passages.

Syringing the Nose—Nasal Lavage.—A syringe on the Higginson principle alone should be used for nasal lavage, and the Wyatt Wingrave or Mayer and Phelps pattern can be recommended. (Fig. 70.)

Sniffing liquid up into the nose is dangerous, as the Eustachian tubes are opened thereby, and the fluid is apt to find its way into the middle ear.

Douching the nose from a raised reservoir of fluid is harmless but ineffective, as the pressure is insufficient to dislodge sticky mucus. Douching by means of the nose-boat is equally futile.

The plain glass syringe of the shops is better than these, but it is much less effective than the Higginson pattern.

Method.—A large and a small bowl or basin are required. In the small one the solution, which should be fairly warm (100° F.), is placed, together with the end of the syringe. The patient stands or (better) sits with head held forward over the larger basin. The nozzle of the syringe is inserted into one side of the nose, being laid along the floor of the vestibule, and not so far in as to come into contact with the sensitive nasal mucosa. The stream is directed first straight back along the floor of the nose, and later upwards and backwards towards the ethmoidal region. During syringing, the patient keeps the mouth well open, and must not raise his head. After one side has been syringed, the nozzle is transferred to the opposite vestibule in order to syringe the other side.

The patient should be taught to do this for himself; he is quickly warned by pain if he syringes too vigorously, or if the fluid finds its way into some recess under too high pressure. After syringing, the nose should be freely blown ***with open nostril.***

The syringe should be sterilized after use, and hung up by its end.

Constitutional Treatment.—The folk-medicine of the world is rich in remedies for " a bad cold," and they have for the most part one common principle, and that is free perspiration.

A hot bath, a hot drink, and a retirement between blankets in company with one or two hot water bottles without any other treatment, is enough to turn a bad cold into a mild one. After the perspiration has continued for a few hours, the patient should be rubbed dry, and clothed in dry flannels. Otherwise, a chill may follow the sweat, and while it is true that acute catarrh is not caused by cold, it is, nevertheless, also true that, once it has set in, a chill, or exposure to cold, like anxiety and other depressing circumstances, will aggravate its symptoms.

The treatment of the other forms of catarrh is symptomatic, but the removal of the cause in secondary acute catarrh must not be omitted.

MEMBRANOUS RHINITIS

Rhinitis with watery discharge, nasal obstruction, and the formation of false membrane may be (1) diphtheritic or (2) non-diphtheritic.

Nasal Diphtheria we have already considered. (See p. 38.)

Non-Diphtheritic (Simple or Septic) Membranous Rhinitis is not uncommon. The infection is usually pneumococcic, but the other pyogenic organisms may also induce the formation of false membrane in the nose.

Symptoms.—The constitutional symptoms are mild, and the patient's usual complaint is stuffiness in the nose with serous or purulent discharge. On examination, the membrane, usually fawn-coloured and fairly tough, is easily seen, being especially conspicuous on the septum.

The *diagnosis* from diphtheria depends almost entirely on the bacteriological findings, but if the patient has a raised temperature, the membranous rhinitis is probably non-diphtheritic.

A membranous rhinitis sometimes accompanies tertiary syphilitic lesions in the nose, affecting the mucous membrane in the neighbourhood of the gummatous deposit or ulcer.

CHRONIC RHINITIS

Two varieties of chronic rhinitis are distinguished.

1. Simple chronic rhinitis.
2. Hypertrophic rhinitis.

(We exclude rhinitis sicca and atrophic rhinitis from this category.)

Simple Chronic Rhinitis arises from frequently recurring attacks of acute rhinitis; from constant exposure to cold winds as in motor-driving; from irritating vapours and dust; from excessive tobacco-smoking and inhaling; and possibly from constitutional disorders such as gout and rheumatism.

It is practically always present in cases of adenoids, in nasal sinus suppuration, and it is predisposed to and perpetuated by obstructing deflections and spurs of the nasal septum.

Symptoms.—Nasal obstruction is the chief complaint and it is generally of the alternating type; that is to say, when one side of the nose is blocked, the other is free. In the lateral recumbent position, the lower nostril is always that which becomes blocked.

On examination, the inferior turbinals may appear to be normal, with nothing visible to account for the patient's complaint, or they may be a little swollen and congested. On the application of cocaine, however, all the swelling disappears, and the turbinals shrivel up against the side of the nose, so that we can get a clear view straight back to the naso-pharynx. This shrivelling up, which proves that the engorgement of the turbinals is active and not passive—not due to œdema or hypertrophy—serves to distinguish simple chronic rhinitis from hypertrophic rhinitis.

Examination for possible causes of the disease should be made, such as adenoids, a deflected septum, sinus suppuration, and so on.

The *treatment* consists in the removal of the cause, and then the rhinitis will get well of its own accord. If the cause cannot be found, the inferior turbinals may be lightly cauterized with benefit. (See p. 267.)

HYPERTROPHIC RHINITIS

The essential pathological features of this form of rhinitis are a permanent dilatation of the venous sinuses of the inferior turbinal from mucoid degeneration of the unstriped muscle-fibres of the inferior turbinal (Wyatt Wingrave), and a hyperplasia of the submucous tissue. In consequence, the application of cocaine to the turbinal is followed by little or no shrinking.

A further stage in the disease consists in the increase of fibrous tissue at the expense of the venous sinuses, and then the hypertrophied turbinal frequently presents a moriform, mammillated, or even papilliform appearance, especially on its posterior portion, when the post-rhinoscopic picture of the large greyish mulberry-looking posterior end is very characteristic. The mucous covering of the middle turbinal bone often participates in the hypertrophy, and by a further increase through the adsorption of water frequently becomes transformed into true *mucous polypi.* (See below, p. 270.) The swelling of this body in the narrow space of the middle meatus may give rise to severe pain at the root and bridge of the nose.

The mucous membrane of the turbinals is frequently much paler than normal, and the mucous secretion is thick and tenacious, giving rise to much hawking and spitting when it passes into the naso-pharynx.

The constant nasal obstruction, frequent frontal headache and fullness in the head, with, perhaps, tinnitus and a degree of deafness from concomitant Eustachian obstruction, occasion the patient much annoyance. Reflex disturbances, such as cough, asthma, bronchitis, and the familiar train of maladies which attend upon nasal obstruction, are frequent results of this troublesome condition.

Any form of simple rhinitis, when long-continued, is liable to end in hypertrophic rhinitis, and the same is true of vaso-motor rhinorrhœa. (See p. 303.) Above all, it should be remembered that the irritation produced by the constant presence of pus in the nose from a suppurating accessory sinus is a frequent cause of hypertrophic and œdematous changes in both turbinals. On the other hand, in hypertrophic rhinitis with its œdematous swellings and polypus formation, we have a condition which interferes to a great extent with the normal drainage of the accessory sinuses, and which, therefore, is liable to lead to purulent sinusitis of one or more of these cavities. While this is true, however, it is also true that many cases suffer from hypertrophic rhinitis with polypus formation lasting for years without any purulent sinusitis developing.

A considerable number of such cases of hypertrophic rhinitis are people who eat and drink too much; or who smoke excessively, or who use snuff; as well as men exposed to rigours of weather, such as locomotive engine-drivers, motor-drivers, and country doctors.

Climate influences its occurrence. It is very common in Scotland, Canada, and the Northern parts of the United States. [illegible]

It is to be noted that there is no pus secreted from the general mucous membrane of the nose in simple or in hypertrophic rhinitis. If, therefore, pus is present in the nasal discharges, there must be some focus of suppuration, generally in a sinus, for which a search ought to be made.

Deviations of the nasal septum, with or without spurs, causing unilateral obstruction, are generally accompanied by hypertrophy of the inferior turbinal on the concave side; mucous polypi are also common effects of the deviation, but they usually appear first, and occur most plentifully on the convex side of the septum. (See also p. 271.)

Prognosis.—If the cause can be removed, and the bulk of the turbinals artificially reduced, cure may be promised. But the secondary effects of hypertrophic rhinitis, such as catarrhal deafness, asthma, and bronchitis, are apt to persist in spite of the removal of the nasal disease.

Untreated, or inefficiently treated, hypertrophic rhinitis passes gradually from bad to worse, and may actually introduce maladies which cut life short.

Treatment.—First of all, the cause or causes of the disease must be sought for and removed, while at the same time the hypertrophies causing the nasal obstruction and interference with sinus drainage are reduced or removed. This entails, first, the reduction in size to its normal dimensions of the hypertrophied inferior turbinal; and secondly, if polypi are present, these must be radically cured.

The septum, if deflected, is straightened (see p. 240); adenoids are removed; suppurating sinuses are drained; and the patient's life and habits regulated.

Reduction in the size of the inferior turbinal may be secured in the slighter cases by cauterization, but the more fully developed hypertrophies will require operative removal.

Cauterization of the Inferior Turbinal.—That part which is hypertrophied alone should be cauterized, and as little damage as possible should be done to the mucous surface of the turbinal. Cauterization, as a matter of fact, is much less frequently performed than it used to be, chiefly because its benefits are rather evanescent. The galvano-cautery alone is recommended.

Method.—The turbinal is anæsthetized by means of cocaine-adrenalin solution on cotton-wool. (See p. 225.)

With a fine, sharp knife, a puncture is made into the most bulbous and fleshy part of the anterior end of the turbinal. A sharp-pointed sterilized galvano-cautery terminal is selected, and passed cold into the puncture just made. With the current on, it is then carried as far back as is necessary, being kept under the mucosa throughout. Finally, it is withdrawn by the same route as it was inserted. Contact both with mucous membrane and with bone should be avoided, as a surface sore will follow the former, and some bony necrosis may follow the latter.

The puncture opening is now touched with a cotton-wool tipped probe dipped in pure phenol. Finally, a dressing is applied and retained for a couple of hours, of a cotton-wool swab, moistened in Sol. phenazon (10 per cent. in water), in order to restrain the reaction.

(The usual method of searing the open surface of the turbinal with the incandescent terminal is not recommended, as it destroys unnecessarily a large area of the ciliated mucous membrane.)

Both inferior turbinals may be cauterized at the same sitting, but as the reactionary inflammation often leads to a blockage of the nose, it is less trying for the patient to have only one side done at a time.

Cautions.—Do not cauterize the posterior end, as middle ear suppuration may follow.

Avoid contact with the septum, as that may lead to a cicatricial adhesion between the septum and turbinal (synechia).

No attempt should ever be made to cauterize the middle turbinal.

Results.—The reaction comes on about half-an-hour after the application with a discharge of thin blood-stained serum, and a slight sense of burning. Turbinal swelling follows, and blocks the nasal passage for three or four days, then it gradually subsides, leaving the nose much more patent than before.

The turbinal, if inspected, is now seen to present a furrow corresponding with the track of the heated needle. This in time cicatrizes, and as the cicatrix is adherent to the bone, the turbinal will remain small for a considerable period. A re-application in a year or eighteen months is, however, usually required, as the effect of the cauterizing on the turbinal turgescence passes off.

Operative Reduction of Hypertrophy of the Inferior Turbinal (Inferior Turbinotomy).

The preparation for these operations are the same as for the submucous resection of the septum. (See p. 226.)

The same caution must be observed as in cauterizing, not to remove any more of the turbinal body than is really necessary to provide sufficient airway. Otherwise, its physiological value being reduced, the evil of a too patent nasal passage will be substituted for the evil of a blocked airway. On the other hand, no hesitation need be felt in sacrificing redundancies and hypertrophies. Physiologically, the most important parts of the inferior turbinal are its anterior and its posterior ends.

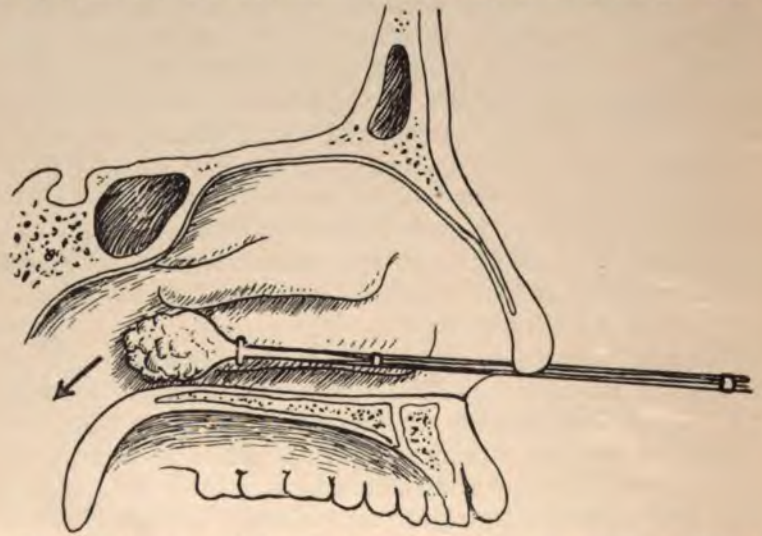


FIG. 89.—Removal of Enlarged Posterior end of the Inferior Turbinal by snaring. After the snare has been fully tightened the bulbous end of the turbinal is detached by a jerk backwards in the direction of the arrow.

(a) *Anterior Turbinotomy.*—Under cocaine-adrenalin paste anæsthesia, the anterior end at its junction with the lateral wall of the nose is snipped through with a pair of nasal shears as far as, but not into, the bone.

A nasal snare is now inserted, the end of the shaft being passed into the incision, and the wire loop being round the lower border of the turbinal. In this position it is tightened, and the soft tissue is thus snared off.

(b) *Posterior Turbinotomy.*—(Removal of Posterior Ends of Inferior Turbinal.)

Posterior turbinotomy is performed when the posterior ends have undergone mulberry hypertrophy, and are causing obstruction, or are leading to secondary changes in the Eustachian tube or middle ear. The presence of the hypertrophy is readily diagnosed on posterior rhinoscopy, when that method is practicable. Otherwise it is visible in the nasal endoscope. (See Plate II., Fig. 4.)

Removal may be effected by the snare or by posterior turbinotomy forceps, and either under local or nitrous oxide anæsthesia—the latter is preferable, as it permits of the finger being passed into the naso-pharynx to guide the snare-loop into place.

Anæsthesia.—*Local,* by tampons soaked in sol. cocaine (10 per cent.) and sol. adrenalin (1-1000), or *general,* nitrous oxide or ethyl chloride being sufficient.

The snare-loop is bent towards the side to be operated on, and passed along the floor of the nose to the pharynx. It is then turned so that the bent loop may engage the posterior end, and when it is felt to do so, the loop is slowly tightened and closed. In manœuvring the loop round the turbinal, the left forefinger in the naso-pharynx is very useful, but when the hypertrophy is large, the loop effects a hold without any such assistance.

When the loop has been drawn as tight as possible, the snare should be pushed *back* into the naso-pharynx so as to sever the posterior end *only.* If it is pulled forward, and the mucosa is tough, the whole of the turbinal covering may be stripped from behind forward. (See Fig. 89.)

If the forceps are used, the posterior end is merely cut through and dropped or twisted off.

Other hypertrophies of the inferior turbinal, of the pseudo-papillomatous and mamilliform nature may be snared in like manner.

After-Treatment.—The slow tightening of the snare prevents serious hæmorrhage, but the patient should be kept in bed for a day or two after the operation. The nose may be syringed through with normal saline the day after the operation, to empty it of blood-clot ; but unless pus is present, regular douching or syringing is not required.

MUCOUS POLYPUS OF THE NOSE

(See also Ethmoidal Suppuration, p. 338.)

The word " polypus " has no precise pathological significance, but it is still retained as a term of convenience because it connotes certain well-recognized and common pedunculated or fimbriated outgrowths of mucous membrane, such as the " mucous polypus " of the nose, the rectum, the uterus, and so on, which modern pathology has taught us are not new growths. In the past, however, the name was also applied to pedunculated malignant growths in the nose and elsewhere, and indeed we still occasionally speak of " malignant polypus," but as a matter of fact, there is a general tendency nowadays to refrain from calling malignant growths, or other tumour formations, "polypus," and to restrict the use of the term to the "mucous polypus," from whatever cause arising.

And this tendency is to be encouraged, since there is no doubt that a simple, or nasal mucous polypus is not a *myxoma*, which is a tumour composed of mucous tissue like Wharton's jelly. The mucous polypus is not, indeed, a new growth at all. It is merely a result of œdema, and the fact that the œdematous tissue adopts the shape of a polypus, simulating in outward appearance a true neoplasm, is a pure accident dependent upon certain local peculiarities.

In other words, the " *mucous*," or to give it its correct pathological title, the " *scrœdematous polypus* " of the nose is merely an expression of inflammation, and is no more a tumour than is the rounded dorsum of the œdematous hand in tenosynovitis of the flexor tendons in the palm.

That being so, we discuss nasal polypi at this point instead of in the section dealing with tumours of the nose, but we must devote a special section to their consideration, because their presence, bringing with it certain adverse mechanical factors, demands special therapeutic measures in addition to those directed to the disease or diseases of which they are the outcome.

The mucous polyp of the nose is composed of loose œdematous connective tissue containing mucigen, with a small number of mucous glands, and is covered with the columnar ciliated epithelium of the nose, save when the polyp is exposed to air sufficiently to dry it, in which case the epithelium loses its cilia and becomes stratified. The same circumstance—exposure to an air-current—if prolonged, may also lead to a fibrosis, a solidification, so to speak, of the semi-fluid interior of the polypus, which then comes to possess a fibro-œdematous structure. But it is not a fibroma.

Any inflammatory condition, if sufficiently prolonged, will lead to polypus formation in certain definite regions of the nose. They sprout from the surfaces of the middle turbinal, and of the hiatus semilunaris; and, less frequently, from the roof of the nose. They arise also from the mucous lining of the accessory sinuses, those from the ethmoidal cells easily finding access to the nasal chamber, where the free space enables them to attain considerable dimensions. When they grow from or near the orifice of the antrum or sphenoidal sinus also, they may appear in the nose. But those which spring up from the lining inside those cavities, and from the frontal sinus, may grow to fill the whole of the interior of their cavity, but they do not reach the nose, and their growth does not seem—unlike true tumour growth within these sinuses—to cause any expansion of the sinus cavities.

In the ethmoidal region, however, free polypus growth does induce serious pressure effects. If the middle turbinal does not itself participate in the polypus-formation, it is not unusual to find it flattened out as thin as a leaf against the bony septum. And when it does participate, the polypus growth comes in time to effect a complete disruption and disintegration of the structure of the bone, and the same remark applies to the whole of the ethmoidal labyrinth, the bone of which, being thin and papyraceous, is evidently unable to resist the swelling of its contained mucous cavities, as the stouter sphenoidal and frontal bones do.

In this condition also, it is not uncommon to find that the bridge of the nose has become broadened as a result of the expanding pressure of the increasingly bulky polypoid masses inside the nose.

It is interesting and remarkable to find that although in such cases of luxuriant polypus formation, the nasal septum participates in the œdema, yet its mucosa very rarely becomes polypoid. All that is produced is a general soft swelling, pitting on pressure, and limited to the ethmoidal septum. The cartilaginous and vomerine portions either escape entirely, or their swelling is relatively insignificant.

Arising in this way, as a consequence of inflammatory, or to speak more accurately, *hyperæmic* conditions, nasal polypi are found in

1. Chronic hypertrophic rhinitis—being analogous to the pseudo-papilloma and solid hypertrophies of the inferior turbinal.
2. Suppurative disease of the ethmoidal cells and other accessory sinuses.

They also form in the disease known as

3. Paroxysmal rhinorrhœa, obviously from the general nasal œdema present in that condition.
4. Further, they appear also not infrequently when the ethmoidal region is the seat of malignant disease, as a consequence of the associated inflammatory reaction.

Polypi vary in number, in size, in rate of growth, and in rapidity of formation, and, when removed, of recurrence, those which are associated with purulent sinusitis being the most troublesome.

The *symptoms* are those of varying degrees of nasal obstruction, generally of gradual onset, though to some patients the obstruction may seem to have appeared suddenly. Occasionally the patient has the sense of a loose body in the nose. In marked obstruction, the voice is toneless, and the sense of smell is lost. The discharge from the nose is thin and watery in catarrhal cases, or thick and purulent when the polypi are secondary to sinus suppuration. The pressure of a large number of polypi, by blocking the orifices of sinuses previously healthy, may induce sinus disease, while epiphora also may arise from obstruction of the nasal duct. As we have already seen, in long-standing cases the distension of the middle meatus may broaden the nose externally. Neuroses, such as paroxysmal sneezing, asthma, cough, etc., are frequent concomitants of nasal polypi, and obstructive deafness is common in this, as in all forms of nasal obstruction associated with inflammatory change.

On *examination*, the cause of the symptoms is, as a rule, easily found in the smooth, glistening, greyish-blue, semi-translucent, movable tumours. Occasionally, only one solitary polypus is present, in which case it may attain to a giant size. But generally speaking, they are multiple and also bilateral. In early cases, we may find only one or two small polypoid swellings on the under surface of the middle turbinal, or between that structure and the lateral wall of the nose, but later the whole of the turbinal may be transformed into a mass of polypi filling up the whole of the middle meatus, and indeed, extending to occupy the entire free space of the nasal cavity.

Such polypi are easily seen, and indeed may actually be found to be protruding from the anterior nares. But when single and in remote localities, they may escape detection, as when they are growing from the posterior end of the middle turbinal, or in the case of small polypi attached to the lip of the accessory opening of the maxillary antrum, or protruding from the sphenoidal sinus. When these last become larger, however, they appear in the naso-pharynx (Choanal Polypi, see below). Such deep-seated polypi may be rendered visible by reducing the turbinals with cocaine; or the nasal endoscope, or posterior rhinoscopy may reveal their presence. Sometimes the patient, by blowing the nose hard, can bring a deep-seated polyp into view.

Choanal Polypus (Fig. 93).—A polypus appearing in the posterior choana usually proceeds from near the lip of the accessory opening of the antrum (antro-nasal polypus), less often from the sphenoidal sinus, or from the posterior ethmoidal region. Polypi from these sites grow back towards the posterior choana, and if neglected, will pass into the naso-pharynx, and appear from behind the soft palate, where they may be seen on looking at the throat. In this situation, the antro-nasal polyp is liable to slough, as its stalk is sharply bent against the edge of the antral orifice.

Diagnosis.—The beginner always mistakes the inferior turbinal for a mucous polyp, and yet the only resemblance between them is that both are conspicuous bodies in the nose. The turbinal is red, the polyp is grey-blue. The turbinal is fixed; the polyp is movable. The turbinal is sensitive; the polyp is devoid of all sensation.

In a case with nasal polypi, it is often necessary to remove the growths before a diagnosis of their cause can be made, but, on the other hand, a true diagnosis is just as often possible at first sight. If pus is seen freely exuding from among the growths; if they are multiple; and if they are recurrent, the cause is sinus suppuration, and our next step will be to determine which sinus or sinuses are affected. (See p. 314.)

If no pus can be seen; if the polypi are few in number, and of slow growth or tardy recurrence, the cause is probably catarrhal rhinitis, but we must be careful to exclude the sinuses before forming a definite opinion on this point.

If no pus can be seen, and if the history of the case is that of violent and prolonged sneezing with profuse watery discharge from the nose, the cause is probably vaso-motor paroxysmal rhinorrhœa. (See p. 303.)

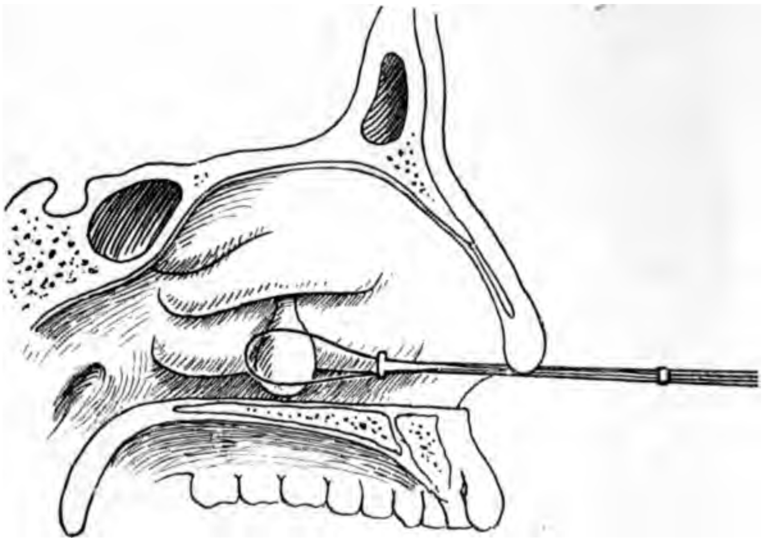


FIG. 90.—Removal of a Solitary Polypus by Snaring. Introduction of the Snare.

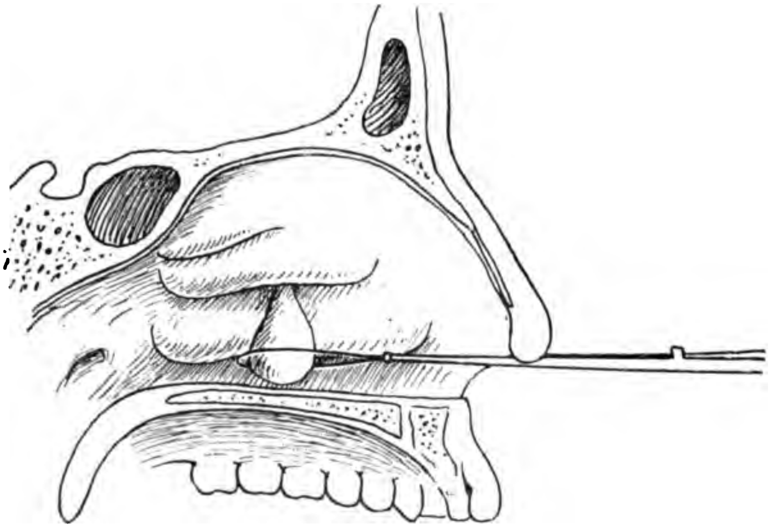


FIG. 91.—Removal of Solitary Polypus by Snaring. The Loop of the snare engaging the Polypus.

If there is a history of profuse epistaxis, with pain and fœtid discharge in one side of the nose, the suspicion of cancer will arise (See p. 292.)

Prognosis.—Naturally, the prognosis is that of the cause of the polypi. As regards the growths themselves, the prognosis is best in the case of solitary polypi without sinus suppuration and of slow growth, and worst in the case of polypi from sinus suppuration. Under modern methods of treatment, however, we can promise non-recurrence and cure, save, of course, in the case of polypi due to malignant disease.

The *Treatment* of nasal polypus resolves itself into two parts; first, the removal of the polypi as radically as possible; and secondly, the treatment of their cause.

The radical removal of the polypoid growths generally involves the removal of the site from which they are growing, and this works out in practice as the removal of the diseased and broken up ethmoidal cells and bone generally by the operation of *curetting.* Before proceeding to curette, however, it is advisable to clear away as many of the bulkier polypi as possible with snare or forceps, or both, for two reasons; first, to restore nasal patency; and secondly, to clear the ground for the operation of curettage.

Moreover, we must not omit to note that in the case of solitary and slow-growing polypi, due to non-purulent catarrh, their simple removal by the snare, if complete, and especially if the bone at the base is also detached, will, in many cases, be sufficient for cure.

Snaring a Polypus.—Local anæsthesia by tampons is employed, and the procedure is almost painless. Heath's modification of Wilde's snare is recommended, with the writer's simple wire-holes. (Fig. 92.)

The cocaine-adrenalin tampons having been removed, the snare-loop is passed along the inferior meatus of the nose, the loop being borne sideways so as to avoid the inferior turbinal. (Fig. 90.) When it has come to a position immediately under the polyp, it is raised upwards under inspection, in such a way that the polyp is received in the wire-loop. (Fig. 91.) The loop is now carried up as far as the attachment of the growth in the middle meatus, and is closed tight, so as to snare the polyp quite close to its attachment.

The snare is not intended to cut through the polyp, so that whenever the growth is felt to be closely enough strangled, it is *pulled* off its attachment to the turbinal with a quick jerk of the hand. This manœuvre may be repeated with fresh snares as often as is required to remove all visible polypi, but it is most suited to cases with single or small polypi. Where they are numerous and large, it saves time and is no more painful to remove them wholesale by means of the author's nasal forceps (Fig 94.)

In nervous patients, with multiple polypi on both sides, two or more *séances* may be necessary to complete the removal.

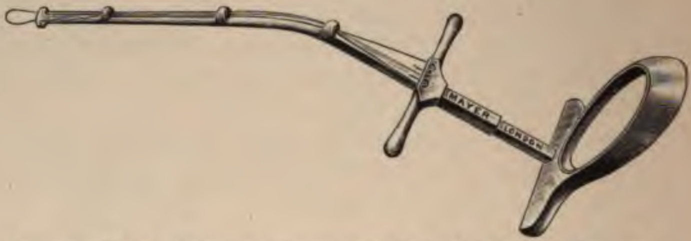


FIG. 92.—Nasal Polypus Snare, with Author's Simple Wire-holes. (Wire with No. 7 English gauge.)

The rule is to effect a clearance on one side at least, so as to restore nasal breathing at once as fully as possible.

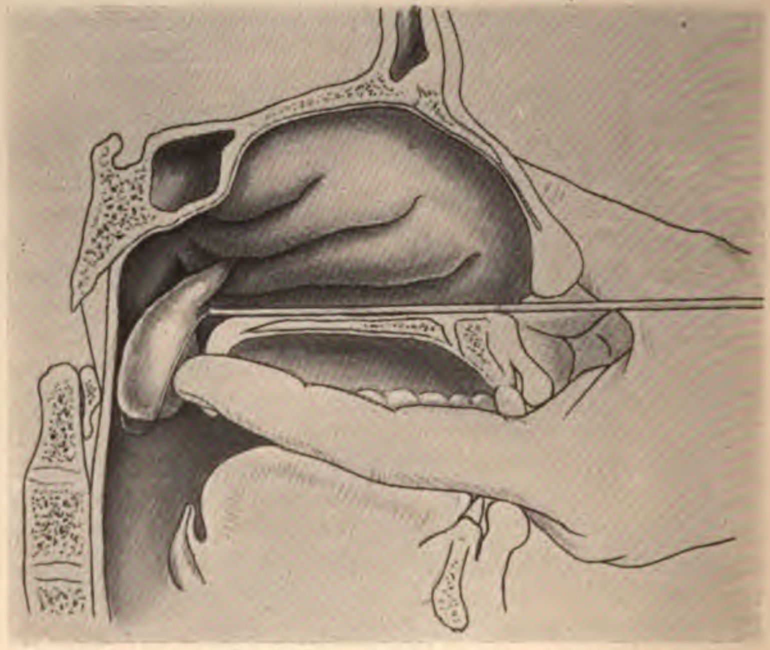


FIG. 93.—Snaring a Choanal (or antro-nasal) Polypus.

Occasionally the patient may prefer a general anæsthetic. (See below.)

The bleeding when polypi are being removed is very free indeed, and preparations must be made for it; but it stops spontaneously, so that packing the nose, which is both unpleasant and liable to induce sepsis, is seldom or never required.

After-Treatment.—If only a single polypus has been removed, the patient may be allowed to return at once to ordinary life and occupation; but if the operation has been extensive, or has caused much bleeding, the patient should be kept in bed for several days.

If further removals are necessary; if a curetting is to take place; or a sinus operated on; or any other nasal operation performed, it is advisable to delay any such fresh interference until at least a fortnight has elapsed from the first operation. Otherwise, we must operate through a septic wound, and the inflammatory reaction may be severe.

In aged patients, the bone of the roof of the nose being very brittle, the removal of polypi should be effected with the most cautious gentleness (Wingrave).

Removal of a Choanal Polypus.—(1) The snare having been inserted into the nose, its loop is guided round the polyp by the finger passed into the naso-pharynx through the mouth. (Fig. 93.)

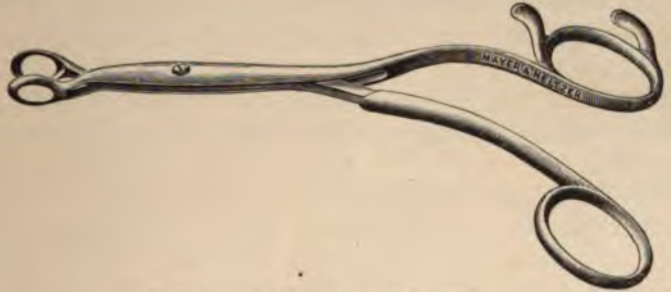


FIG. 94.—Author's Nasal Forceps.

(2) Or the author's nasal forceps (Fig. 94) may be used, being passed into the nose and turned so as to grasp the pedicle of the polypus close to or within its orifice of origin.

(3) Large choanal polypi are most easily removed by means of Jurasz's adenoid forceps (Fig. 134) inserted into the naso-pharynx through the mouth.

For these manipulations a general anæsthetic (nitrous oxide or ethyl chloride) is advisable.

Should an antro-nasal polypus recur after it has once been removed the maxillary antrum should be opened as in the Caldwell-Luc operation (see p. 334) and its mucous lining, if œdematous, removed. But as a matter of fact recurrence after complete removal is rare, as in these cases the antrum is not the seat of suppuration.

CURETTAGE OF THE ETHMOID

for polypoid disease (Lambert Lack).

In multiple ethmoidal suppuration with polypus formation, simple avulsion of the polypoid growths, whether by snare or forceps, is insufficient to cure the case. The diseased areas must be entirely removed, and for this purpose curetting is the safest, most rapid, and most efficient method.

Curettage should, however, be reserved for suitable cases. It is unnecessary when polypi are few and slow-growing, and when there is no ethmoidal cell suppuration. The most suitable cases are those where long-standing and extensive disease has already broken up and destroyed the normal ethmoidal labyrinth.

Anæsthesia.—A general anæsthetic (nitrous oxide and ether, or chloroform) is advised. But the operation, even when the disease is extensive, can be done under local anæsthesia (cocaine-adrenalin tampons) if the patient does not object to two or more *séances.*

The patient may be seated upright in a chair, or may lie on the back with head and shoulders raised, and head turned on one side.

Very free hæmorrhage must be anticipated, for which reason the sitting position brings many advantages. If the patient is reclining, blood is apt to find its way into the air-passages unless a captive sponge is placed in the naso-pharynx.

During nasal operations in the recumbent position attended with much bleeding, it is advisable to turn the patient's head well on the one side in order that the blood may run freely from the nose, and in order that blood coughed up from the throat may find its way into the dependent cheek and not fall back again into the pharynx. If the head is lowered the bleeding will be increased, and with it, the surgeon's embarrassments.

It is often possible, however, even when the patient is recumbent, and without a sponge in the naso-pharynx, to insert a dam of gauze-strips into the back of the nose sufficient to keep the pharynx from being flooded. For the rest, one trusts to rapid throat-sponging, for which at least a dozen George throat-sponges should be at hand, and should be the sole charge of one nurse.

If adrenalin is used at the operation, severe reactionary hæmorrhage is likely to set in after the operation, and may necessitate packing, whereas the ordinary bleeding at the operation comes or is brought to a permanent standstill, before the patient leaves the operating table.

The *preparation* of face and nose is the same as for submucous resection. (See p. 226.)

Operation.—After any additional obstructing polypi have been removed, a ring curette is passed into the nose and borne upwards to the posterior ethmoidal region. Held lightly in the hand, and with the sharp edge turned outwards, the instrument sweeps through the ethmoidal region in a direction downward and forward, removing in its course, polypi, broken up pieces of ethmoidal bone, and débris. The free hæmorrhage prevents any inspection, so the curetting must be done under the guidance of the surgeon's little finger, passed (gloved) into the nose from time to time. It is possible to palpate with the finger the whole ethmoidal area from the sphenoidal to the anterior ethmoidal region, and thus to form a mental picture for the guidance of the curette.

In curetting, only light strokes are to be used, and any bony resistance felt is not to be overcome. If the curette is kept on the outer wall of the nose, the only damage likely to be done is a breaching of the bony inner wall of the orbit with consequent orbital ecchymosis. But even this is avoidable with ordinary care.

Both sides may easily be cleared at the one sitting.

Difficulties and Dangers.—The reader is referred to p. 336 for the anatomy of the ethmoid. The limits of operating there laid down should not be transgressed.

After-Treatment.—Hæmorrhage ceases, as a rule, before the patient emerges from the anæsthetic. If it continues, the ethmoidal region may be tamponned with dry ribbon gauze or gauze strips, the inferior meatus being left clear of packing. The gauze is removed in not more than twelve hours, and not replaced unless severe hæmorrhage is recurring.

The nose may be syringed daily with normal saline in suppurating cases. Otherwise, there is no need to repeat the lavage after the first and second day.

The patient is usually able to get up within a week of the operation.

Results.—After complete curetting, freedom from polypus recurrence is the rule. Occasionally, a curetting has to be repeated once or even twice. But the ultimate result is always cure of the polypus-formation. When the parts have cicatrized, the roof of the nasal cavity assumes a smooth form where the little orifices that lead into the remaining ethmoidal cells are clearly visible.

RHINITIS SICCA AND ATROPHIC RHINITIS

In the following section we are dealing with several nasal disorders which are generally grouped together not because they are pathologically akin, but because they manifest one common clinical characteristic, and that is, a tendency to dryness of the nasal mucosa—what we may venture to term *Rhinoxerasia,* (ξηρασία, dryness).

We include in this group (1) the so-called Rhinitis Sicca, and (2) Atrophic Rhinitis, of the latter of which the following three varieties are distinguished :—

(a) *Simple (non-fœtid) atrophic rhinitis;*
(b) *Syphilitic atrophic rhinitis; and*
(c) *Ozænatous atrophic rhinitis, or ozæna.*

Of these, the last two are certainly specific diseases, and it is equally certain that rhinitis sicca and simple atrophic rhinitis are not diseases at all. They merely represent a physical condition—rhinoxerasia—and are, in other words, the result, it may be the terminal stage, of certain other diseases of the nose.

RHINITIS SICCA

This term is, therefore, a misnomer, since, as we have just said, the condition to which it applies is a physical effect of other diseases, and not a disease-entity in itself.

The one characteristic of the condition is a relative dryness of the nasal mucosa, which is manifested in the mucous secretion being scanty and thick so that it readily dries into viscous threads and adherent scales, particularly in those areas which are normally exposed to the ingoing air-currents, such as the antero-inferior portion of the septum (Little's Area) ; the antero-inferior surface of the middle turbinal ; and—to leave the nose—the posterior wall of the naso-pharynx and pharynx. The condition is one which may be due to an enfeeblement of the ciliary action of the cells of the mucous surfaces in consequence of which the mucus is permitted to cling, to lodge, and finally to dry into a scale or fine crust, a process which, by further impeding the ciliary movement, furnishes a typical instance of the vicious circle.

As predisposing factors, we may mention an unduly wide nasal passage from whatever cause arising, and, especially, the extensive removal of turbinal tissue, seeing that that may both render the nasal passages abnormally roomy, and provide, likewise, areas of non-ciliated cicatricial tissue upon which the mucus readily dries.

Rhinitis sicca is distinguished from simple (non-fœtid) atrophic rhinitis by its crusts being scaly and fine, and by their being composed solely of mucus.

SIMPLE (NON-FŒTID) ATROPHIC RHINITIS

is characterized by the formation of crusts not in one or two areas only, but generally distributed all over the interior of the nose, and these crusts are not only composed of dried mucus, but they also contain pus-cells and epithelial débris, while atrophy of the mucosa, and particularly of the inferior turbinal bone, and sometimes of the septal cartilage, is pronounced.

Respecting this feature of atrophy, it is probably the case that it is the simple effect of the pressure of the adherent crusts. This pressure operates by producing anæmia and atrophy of the structures under the crust, its effects being most strikingly manifested in the fenestration beneath intact mucosa of the septal cartilage in Little's Area. The atrophy, in a word, is mechanical, and the fact that it is more obvious in "atrophic rhinitis" than in "rhinitis sicca," is due to the crust formation in the former being more widespread and constant.

Simple atrophic rhinitis is distinguished from ozænatous rhinitis by its milder course, by the absence of fœtor from its crusts, and, according to Wyatt Wingrave, by the absence of acid-fast bacilli. It is seen in its most advanced and typical form in the later stages of ethmoidal or sphenoidal sinus suppuration, when extensive curetting has been necessary, and when there is still some lingering suppuration.

Treatment (of Rhinitis Sicca and Simple Atrophic Rhinitis). Obviously it is important to avoid producing the conditions which lead to rhinoxerasia, and in operating on the nose, therefore the rule is to remove as little as possible of the physiologically active structures; and of all such structures the most important is the inferior turbinal body. It is also important to avoid the infliction of open wounds and the production of raw surfaces which, on healing, become extensive areas of scar-tissue covered with non-ciliated epithelium.

We may note in passing that, as D. Shaw has pointed out to me, post-operative rhinitis sicca or atrophic rhinitis is less common in the humid climate of the British Isles than it is in more arid countries. Operators abroad, therefore, as in the United States, Canada, India, or Africa, soon learn to be more cautious in removing intranasal tissues than their English colleagues need to be.

For the treatment of the condition once it is started, if it is mild as in *rhinitis sicca*, the irritation and stuffiness it induces may be relieved by the daily use of simple liquid paraffin sprayed into the nose by means of an atomizer.

For the more advanced types (*atrophic rhinitis*), the nose should be syringed daily with the alkaline solution, detailed on p. 262, and in sinus cases, provision should be made for the free drainage of any sinuses that may still be suppurating. For such lingering sinus suppuration, vaccines are often of service. (See p. 371.)

For *syphilitic atrophic rhinitis* see p. 289.

ATROPHIC RHINITIS (OZÆNA. OZÆNATOUS RHINITIS)

Ozænatous rhinitis is a disease the causation of which is unknown. It is clinically characterized by the transformation of the ciliated columnar epithelium of the nose into pavement epithelium; by the presence of crusts and stagnant secretions exhaling a peculiar and repulsive odour; by atrophy of the mucosa and skeleton of the respiratory part of the nose, and frequently, by an extension of the disease, also along the air-passages, to the naso-pharynx, pharynx, larynx and even trachea.

It shows no tendency, however, to leave the route of the respiratory current and to attack the mouth, the laryngo-pharynx, or the œsophagus, and even the middle turbinal and the upper regions of the nose are seldom invaded, the middle turbinal being indeed usually enlarged and œdematous, in striking contrast with the inferior turbinal. Moisture is thus obviously inimical to its spread.

The crusts and secretion in a developed case consist of pus cells, epithelial cells, sticky or dried mucus and a host of bacteria of different kinds, to the influence of which is due the peculiar fœtor characteristic of the disease.

Recent views refer the disease to a special organism, the *cocco-bacillus fœtidus ozænæ* (Perez); or otherwise, would look upon it as a para-tuberculous affection (M'Kenzie, Mackeith, and Wingrave). But the theories of causation of ozænatous rhinitis can hardly be numbered.

Symptoms.—Ozæna attacks females by preference, and it usually begins in childhood between the ages of seven and twelve, but so slow is its progress that it seldom attracts any attention until after puberty, when the crust-formation and fœtor begin to force their presence upon the patient and her friends. The disease is at its height from the age of twenty to thirty years, and then it usually undergoes a slow involution, the crust formation becoming scantier, the nose cleaner of discharge, and the atrophy less noticeable.

A number of cases develop tuberculosis in the lungs or elsewhere, but the nasal disease, nevertheless, seems as a rule to end in recovery.

In the earlier stages, the mucous membrane of the nose is dry and studded with minute, discrete, circular, greyish spots of thin, dried secretion, which even at this period disseminate faint whiffs of the characteristic odour. Later, when it is more fully developed, the patient complains of nasal discharge and crust-formation, but, fortunately for her, she is herself unaware of the odour she exhales, as her anosmia is usually absolute. Herein lies a minor point of distinction from sinus suppuration where the subjective fœtor is often a source of great annoyance to the patient.

On inspection at this period, the most striking feature is the wide roomy nostril, the walls of which are lined with sticky, yellowish crusts adherent to the underlying mucous membrane. These crusts may be removed in great casts or flakes, laying bare an excoriated and roughened surface, over which little bleeding-points and minute specks of pus are to be seen. The sticky secretion may also be festooned across the nasal cavity in strings.

When the nasal passages have been cleared of crusts and secretion, the roominess of the nostril is seen to be caused by a marked atrophy of the mucous surfaces and especially of the inferior turbinal body, which may seem to have so completely disappeared that we can see straight back into the naso-pharynx.

A flat nose with gaping nostrils is typically associated with ozæna, and some observers look upon this type of nose as a predisposing cause of the disease, but it is probably only an effect, as is also the dimpling of the bridge to which we have already alluded. (See p. 214.)

When the septum is strongly deviated, the disease falls with more severity upon the wide than upon the stenosed side, but the latter is not spared, however.

Crust deposit and desiccation of the mucous surface may also be seen in the posterior pharyngeal wall, in the cavity of the larynx, and even in the trachea.

Generally, the patient is anæmic and unhealthy looking.

Nasal Obstruction.—In addition to the usual symptoms of discharge and crust formation a complaint of obstruction to nasal respiration is not infrequently made by patients with advanced atrophic rhinitis, although, as a matter of fact, the nasal passages are found to be much more roomy than in the normal nose. This curious symptom, which is, of course, an erroneous observation on the patient's part, is probably due to a combination of factors. First of all, the tactile sensibility of the nasal mucosa being diminished, the patient no longer experiences the normal sensation produced by the air-currents passing in and out through the nose. Secondly, the unduly wide passages permit of an inspiration and expiration so free from muscular effort that the respiratory kinæsthetic sensation is lowered, so that the act of respiration makes less impression upon the consciousness than when the nose is normal or the seat of real obstruction. Thirdly, when the patient attempts to blow the nose, the air rushes through the widely dilated channels so suddenly and impetuously that the soft palate flaps upward into the naso-pharynx like a tongue-valve, and so prevents or seriously hinders the expiratory blast from sweeping forcibly through the nasal passages.

Paradoxical as it may seem, therefore, a certain amount of impediment to the airway in the nose is necessary, both for comfortable nasal breathing and for clearing the nose of its secretions by blowing; and the absence of this normal impediment in advanced atrophy gives rise to the belief in the patient's mind that she is unable to breathe through the nose.

Diagnosis.—In *syphilis* affecting the nose, there is often crust formation and fœtor, but the odour is different from that of atrophic rhinitis, and careful examination will reveal in syphilis a gummatous ulcer affecting the osseous parts of the nose, where bare bone may be felt on probing. In atrophic rhinitis (ozæna) there is no necrosis or caries of bone, although an undoubted osseous atrophy exists. From the atrophic rhinitis which follows syphilis of the nose the distinction may be made as outlined on p. 289.

From *rhinitis sicca*, even in its most aggravated forms, ozænatous rhinitis is differentiated by the odour, by the tendency to dense crust-formation, and according to Wyatt Wingrave, by the presence in the crusts of acid-fast bacillary forms in large numbers.

Complications.—Apart from atrophic pharyngitis, laryngitis, and tracheitis, ozæna is not uncommonly associated with an intractable form of conjunctivitis. And we have already mentioned the liability to tuberculosis, a fact which renders periodic examination of the lungs and sputum necessary.

The nasal disease occasionally sets up accessory sinus suppuration, generally in the antrum and ethmoidal cells. But the contention of Grünwald that ozæna is always caused by accessory sinus suppuration is no longer credited.

The *treatment* of ozæna is symptomatic, and consists in the adoption of measures to remove discharge and crusts, and to prevent and hinder their re-accumulation, but it is possible that even such simple cleansing measures have also a curative action, in that they wash away from the surface of the mucous membrane the material which produces the disease.

The author advises the use of glycerine tampons—pledgets of cotton wool soaked in glycerine, and inserted by the patient into each side of the nose for twenty minutes daily. The tampon should be large enough to fill the nose, and to extend along its whole antero-posterior length. The glycerine causes a free exudation of serum and watery mucus, which continues for some time after the tampon is withdrawn, and this loosens the crusts and permits of their being blown or syringed out. As a matter of fact, when the treatment is in daily progress, crusts seldom form. Should they do so, they will appear in regions which escape the daily action of the glycerine, and it will be generally found that the glycerine tampons are being improperly inserted, or are of insufficient size. Under this treatment, the submucous tissue regains bulk, and the atrophied turbinals gradually assume something of their normal shape and fullness.

In addition, or as an alternative, if the glycerine is badly tolerated, the nose should be regularly syringed with :

℞. P. Potass. Chlor.	
P. Sod. Bibor.	
P. Potass. Bicarb.	*aa.* ʒiv.
P. Sacch. Alb.	℥i.—M.

Sig. ʒii in half-a-pint of warm water. Or Dobell's Solution may be used. It is

℞. P. Sod. Bicarb.,	gr. 100,
P. Sod. Bibor.,	gr. 100,
Ac. Carbol.	m. 100,
Aq. ad.	Oii.—M.

Many other remedies are at the practitioner's disposal in this obstinate malady, but nearly all owe what good they impart to a combination of the physical stimulation of the mucosa and the washing away of crusts and impurities.

A *vaccine treatment* by the Perez bacillus has also been tried and is sometimes advocated.

Operative measures inside the nose should be avoided. The swollen and œdematous middle turbinal is best left alone. If the antrum is full of pus, it should be opened intranasally, but extensive operation should be avoided. Adenoids and tonsils should *not* be removed, as the natural tendency is for the disease to induce an involution and disappearance of all lymphoid tissue about the nose and throat.

Rhinitis Caseosa is a rare condition characterized by the presence of a foul-smelling cheesy mass in the nose, always associated, it is said, with sinus disease. When in great bulk, nasal and facial deformity may be produced. Sometimes it contains a foreign body or a rhinolith as a nucleus. It causes a foul discharge and induces fœtor, both subjective and objective.

Treatment.—A small collection may be expelled by syringing, but if large a general anæsthetic will be required in order that the mass may be broken up and scooped out by curettes and sharp spoons.

SYPHILIS OF THE NOSE

Congenital Syphilis.—The classical symptom of congenital syphilis in infancy is " snuffles," a name which is applied to a chronic nasal catarrh with noisy breathing and an acrid discharge of muco-pus excoriating the upper lip. On inspection, areas of congestion with mucous patches, usually on the septum, may be seen. Necrosis of bone or cartilage does not occur at this stage, but at or about puberty a necrosis of the cartilaginous or bony septum may occur, producing serious deformity if not promptly checked by suitable remedies.

Acquired Syphilis.—*The Primary Sore* sometimes forms on an ala or on the antero-inferior part of the septum from digital infection.

It appears as an indurated or nodular swelling which undergoes ulceration. The glands enlarged in consequence of chancre in this position are the submental, the submaxillary and the pre-auricular. They are hard and indolent.

Diagnosis.—From epithelioma it is distinguished by its short history, and by its tendency to spontaneous recovery in a few weeks. It may also be mistaken for a lupus deposit, although lupus is much more chronic, and is attended with less induration.

Treatment.—As soon as the diagnosis is made, constitutional treatment should at once be begun. (See p. 71.) Locally, a mild antiseptic ointment, such as Ungt. ac. boric, is all that is required.

The nasal symptoms of **secondary syphilis** do not call for much comment. A slight nasal catarrh, with one or two mucous patches, may coincide with the secondary phenomena of the disease in the pharynx and on the skin.

It is very different with regard to **tertiary syphilis**, for the nose is at times the seat of " one of the most destructive forms of phagedæna." Gummata may form in the septum, in the turbinal bodies, or on the floor of the nose. At first they appear as firm, circumscribed, reddish swellings, but they soon become ulcerated, and extensive necrosis and destruction of the osseous tissue and soft parts may proceed with extraordinary rapidity. The nasal fossæ on examination will be found occupied with crusts and foul discharge, and careful probing will generally reveal necrosed bone. Wingrave has observed that the spirochæta pallida exists in large numbers in smears and scrapings from tertiary lesions of the nose. The patient disseminates a most sickening and penetrating fœtor. When the disease attacks the upper parts towards the roof of the nose, it may be impossible, at first, to estimate the extent of the necrosis, but probing and other interference in this region must be avoided, as the cranial cavity may be broken into, and septic meningitis set up. When this region is attacked, swelling, tenderness, and pain over the bridge of the nose appear, and after the extrusion or removal of the sequestra great deformity from collapse of the bridge is to be expected.

Gummatous deposit on the septum or turbinal bodies we seldom have an opportunity of seeing in its first stage, as no symptoms are produced until ulceration sets in with its discharge of blood and pus. Even nasal obstruction does not seem to attract any attention until after ulceration.

Such cases, when examined, may show very little wrong with the nose at first sight. All that is visible is complete stenosis of the affected side of the nose from swelling of the septum and the lateral wall, the mucous membrane of which seems to be neither infiltrated nor ulcerated, and yet the application of cocaine has no effect upon the tumefaction. The stenosis remains. If a probe be now passed into the nose beyond the stenosed area, dead bone may be felt, and, in any case, bleeding from the wounding of granulations ensues.

On the floor of the nose, a similar blockage may be produced, but as a rule the stenosis is less complete, so that the site of the lesion is visible, with granulations sprouting from the floor of the nose. This is the upper end of a perforating gumma of the bony palate, the lower end of which will be visible in the mouth.

We have already alluded to syphilis as it affects the alæ and vestibule. (See p. 257.)

Progress.—Syphilis of the nose is serious not only by reason of the deformity it may cause. It is also dangerous to life by reason of the facts, first, that the bone attacked lies in contact with the meninges of the brain, and secondly, that when the bony necrosis is extensive, the large open bone wound becomes septic and may lead to death from septicæmia. In such circumstances, also, even the most vigorous constitutional anti-syphilitic treatment may be impotent to bring about cure. The milder cases respond promptly, however, and when the dead bone has been exfoliated and removed, they soon get well, frequently leaving a legacy, however, in the shape of nasal deformity, or what is known as syphilitic atrophic rhinitis.

Syphilitic Deformities of the Nose.—The whole of the external nose may be destroyed, leaving a mere hole. But as a rule, the deformity affects either the upper or the lower half of the feature only. The former, the bony bridge, is destroyed, or what amounts to the same deformity, it sinks in, as a result of syphilis of the nasal bones and ethmoidal septum. The point and lower half become depressed so as to give the nose the appearance of a broken beak, when gummatous ulceration attacking the alæ and columella undergoes healing.

I have also drawn attention to a curious absorption of the bone of the premaxilla with loss of the upper incisor teeth and sinking in of the upper lip as a consequence of necrosing syphilis of the bony septum.

Syphilitic deformity of the nose is produced during the period of healing, and it is therefore obviously due not only to the actual destruction of the framework of the organ, but also to the effect of cicatricial contraction. The premaxillary deformity, however, was noted as having occurred during the active stage of the nasal disease.

Syphilitic Atrophic Rhinitis is an extreme form of rhinitis sicca, and appears when extensive gummatous disease has destroyed large areas of the ciliated nasal epithelium, and has replaced them with areas of scar-tissue.

In addition to this obvious form, it occurs also in children with congenital syphilis in whom no history of gummatous disease can be obtained. In such cases it closely resembles ozænatous rhinitis. But the characteristic odour of ozæna is absent, as are the acid-fast bacilli which are found in the other (Wyatt Wingrave). These features, combined with the positive Wassermann reaction, suffice to establish the diagnosis of syphilis.

Treatment of Nasal Syphilis.—If seen before ulceration and bone necrosis takes place prompt and energetic treatment by the salvarsan drugs, coupled with the iodides and mercury, will cut short the development of the disease. (See p. 71.)

If ulceration has taken place, and bare bone can be felt, prompt response to treatment may, nevertheless, be expected if the sequestrum is small, or if the bone is merely perforated, as in a septal or palatal gumma.

These are the cases in which Potass. Iodid. in doses of from 10 grains upwards three times a day is most serviceable. It should, of course, always be associated with mercury.

When osseous necrosis is extensive, and especially when the presence of pyrexia shows the absorption of septic toxines, the response to treatment, even the most energetic, is sometimes slow in appearing, and may, indeed, never appear at all. The patient should then be kept in bed; the nose should be regularly syringed with warm antiseptic lotion; and as soon as the sequestra begin to feel loose, the patient should be anæsthetized, and as much of the dead bone broken up and removed through the natural passages as can be got away. In doing so, it is necessary to refrain from active or forcible removal of sequestra near the roof of the nose, or in the sphenoidal region, as in the effort the dura, which is frequently exposed in these cases, may be torn through with fatal consequences.

For the syphilitic atrophic rhinitis, the salvarsan group of drugs along with potassium iodide, is very serviceable. But in all cases, and at all stages, mercurial treatment should also be employed.

Tuberculosis of the nose, apart from lupus, is rare. When it does occur, it is generally in association with pulmonary tuberculosis, but a primary infection of the septum is described at the spot where simple ulcer appears, and from the same cause, namely, infection by the finger nail. It takes the form of a tuberculous nodule (tuberculoma), or of an ulcer presenting the same features as tuberculous ulcers elsewhere in the upper respiratory tract.

Treatment.—If definitely circumscribed, the tuberculous deposit should be excised as a whole, under cocaine-adrenalin paste anæsthesia, the mucous membrane being elevated from off the smooth surface of the cartilage in a single sheet.

LUPUS OF THE NOSE

1. *Cutaneous.*—As is well known, lupus of the face frequently starts from the nose, the site of origin being inside the ala, or just within the vestibule in the little recess that lies inside the tip of the nose. From this starting-point, the disease spreads outward over the cutaneous surface of the organ.

The first manifestation is the lupus nodule, a shot-like tubercle in the deeper layers of the skin, surrounded by an areola of hyperæmia. As the nodules increase in number, the skin becomes infiltrated with them, and tends to break down to form the lupus ulcer, which spreads in one direction as it heals in another. The granulations of the ulcer are exuberant, the edges are raised, and considerable destruction of tissue may ensue. The cicatrized surfaces are thin and vascular.

2. *Lupus of the Interior of the Nose* is not so common. It also starts in the vestibule, passing on to infect the septum, or the first deposit may appear on the anterior end of the inferior turbinal, or on the floor. When it attacks the septum, perforation is produced, but it does not, like syphilis, perforate the bony septum. Being an indolent disease, it is, as a rule, considerably advanced before the patient seeks relief, and nasal obstruction with perhaps some crust-formation and a little bleeding is all that he complains of.

The extremely slow progress, together with the signs of cicatrization, enables us to distinguish lupus of the nasal mucosa from cancer. Its microscopic characters, and the presence of nodules differentiate it from the much rarer rhinoscleroma.

The interior of the nose in lupus is rough, granular-looking, pale, and dry, with some tendency to crustation.

Treatment.—The treatment of cutaneous lupus by the Finsen light, by X-rays, or by chemical caustics, lies beyond our scope.

Lupus of the interior of the nose, when it is confined to one small area, may be excised like a tuberculoma. Otherwise, the nodules may be destroyed, methodically and in detail, by diathermy or galvano-cautery puncture.

Where the disease has spread to involve the greater part of the interior of the nose, curetting under an anæsthetic is the quickest and most satisfactory way of dealing with it. Scarring and the consequent tendency to closure of the anterior nares may be combated by the wearing of vulcanite tubes at night.

The disease is extremely chronic, and very obstinate, and the treatment must be repeated at regular intervals.

Pfannenstill's method of treating lupus of the nose by sodium iodide internally with hydrogen peroxide tampons to the nose, I have not found to be satisfactory.

Rhinoscleroma consists of a dense chronic infiltration and thickening of the mucous and submucous tissues of the nose attended with rhinoxerasia, and later, with cicatricial tissue formation and contraction. It leads to great obstruction, and may spread to involve the soft palate, pharynx, and larynx. The disease prevails in Poland and Central Europe, but is almost unknown in England. The treatment is symptomatic.

NEOPLASMS OF THE NOSE AND ACCESSORY SINUSES

Innocent growths—indubitably innocent growths—of the interior of the nose are quite rare, but growths of a sarcomatous or endotheliomatous structure in the ethmoidal region, generally reported by pathologists to be malignant but not behaving clinically as such, are not so uncommon. We shall discuss nasal neoplasms, therefore, as in three classes: innocent; locally malignant; and malignant.

INNOCENT NEOPLASMS

Papillomata appear as red, soft, lobulated growths, sessile or pedunculated, generally attached to the lower turbinal or to the septum. They vary in size from a pea to a filbert, and are occasionally multiple. Care should be taken not to mistake for true papilloma the fairly frequent lobulated hypertrophy of the inferior turbinal found in hypertrophic rhinitis (the so-called *pseudo-papilloma*).

Treatment.—The growth should be snared off under local anæsthesia, and the base, after the bleeding is stopped, touched with lunar caustic or the galvano-cautery, to prevent recurrence.

True Fibromata occasionally appear, of very slow growth and firm consistence. Pathologically and clinically they differ from the fibro-angiomata of the naso-pharynx and posterior regions of the nose in being less vascular.

If the tumour is large, a Rouge or lateral rhinotomy operation may be necessary for its removal. (See p. 300.)

Angioma (Bleeding Polypus of the Nose; Bleeding Polypus of the Septum) is perhaps the least uncommon of the innocent neoplasms of the nose. Situated on the septum near its antero-inferior angle, or on the inferior turbinal, it presents itself as a rounded, movable, purplish growth, rather like a small currant, but raw on the surface and bleeding readily when touched. Frequent and prolonged epistaxis is indeed the invariable symptom of its presence.

Treatment.—Imperfect removal causes more bleeding, and is followed by recurrence.

The simplest and most reliable plan is, under cocaine-adrenalin paste anæsthesia to make an incision through the mucosa clear of the growth down to the cartilage or bone in such a way as to encircle the base of the growth. The whole mass can then be raised off the cartilage by means of an elevator and entirely removed without any hæmorrhage. The raw surface left soon heals over.

Osteoma of the ivory type appears as a rarity in the frontal sinus, and giving rise by its growth to pain and swelling, may require removal through an external incision.

Mucocele is a not uncommon disease of the sinuses, usually of the ethmoidal or fronto-ethmoidal, and less commonly of the antrum.

It used to be supposed that a mucocele was produced by atresia of the sinus orifice and retention of the normal mucous secretion in the sinus cavity. But the modern belief is that a mucocele is a mucous cyst (and that in turn is a form of adenoma), which, undergoing active enlargement, leads to a distension of the bony walls of the sinus and their slow atrophy.

Symptoms.—In the frontal and fronto-ethmoidal region, the growth of the cyst leads to the appearance of a smooth, painless, rounded swelling of the roof or inner wall of the orbit. The tumour, in growing, may cause considerable displacement of the globe, but so slow is the rate of growth that the eye accommodates itself to the new position, and diplopia is rare.

The tumour feels tense and elastic on palpation, and crackling of the thinned, expanded bone may be perceptible on pressure.

In the lower ethmoidal region the direction taken by the enlarging cyst leads it towards the interior of the nose, where it appears like a large or cellular middle turbinal.

Treatment.—Mucocele of the *frontal sinus* must be removed through an external incision. The cyst proper is exposed by removing as much of its egg-shell covering as is necessary, and if it can be entirely removed, the wound may be closed up again without breaking down the infundibulum to effect nasal drainage.

In the *fronto-ethmoidal* region, it is often possible to open into the cyst from the nose, removing as much of the sac as will prevent the re-constitution of the cyst. Quite frequently, considerable areas of the orbital wall will be found to have melted away under the pressure atrophy.

In the *lower ethmoidal* region, the diagnosis usually comes as a surprise, when, in the act of removing what seems to be a distended middle turbinal, a cavity is broken into containing a white opaque liquid. As much of the thin bone and sac wall as can be reached should be removed.

LOCALLY MALIGNANT NEOPLASMS

are found usually in the upper ethmoidal region of the nose Microscopically their structure is that of fibro-sarcoma or round-celled sarcoma, with large blood-spaces. In their growth, which is rapid, they erode bony walls, such as the septum nasi, the wall of the orbit, and the nasal bones, and frequently form attachments as they spread to various parts of the nose. They seem to spring most frequently from the cribriform region and grow downwards into the nose, but the cribriform also may be eroded by their growth, and the dura laid bare in the roof of the nose.

This class of tumour resembles the naso-pharyngeal fibroma in its unrestrained local growth, in its tendency to erode bone, and to form connections as it spreads, and also in its vascularity and readiness to bleed profusely. For that reason, in spite of the fact that the naso-pharyngeal fibroma sometimes stops growing spontaneously and undergoes involution while the nasal tumour does not do so, it is possible that these two classes of tumour may be fundamentally akin.

Symptoms.—The earliest symptom is epistaxis, generally profuse and hard to locate on ordinary anterior rhinoscopy. Epistaxis emanating from the ethmoidal regions should always be regarded with suspicion. Later, a watery or muco-purulent and often fœtid discharge appears, and pain may be experienced, although it is not a common symptom.

The tumour first makes its appearance according to the direction it takes. When it grows downward, it appears in the nose as a grey, fleshy, lobulated mass, often movable, and very tender, and bleeding freely when touched.

Or it may erode the inner wall of the orbit, and cause displacement of the eyeball sometimes with diplopia, projecting into the orbit where, on deep pressure, it can be felt as a fleshy swelling.

Again, the nose on rhinoscopy may appear to be normal, and the first appearance of tumour growth be visible on the exterior of the nose, the tumour having made its way through the nasal bone or ascending process of the superior maxilla. In this development it may lead to broadening of the root of the nose.

If allowed to proceed unchecked, the growth causes death either by erosion of the dura and meningitis, or as a result of the continual severe hæmorrhages and consequent anæmia. But if radically removed by operation it may never recur again.

Secondary deposits outside of the nose do not occur.

MALIGNANT NEOPLASMS

In character, malignant growths of the nose belong to the epitheliomatous group of tumours. When the microscopic appearances of the tissue are those of the sarcomatous group, the malignancy, as we have just seen, is usually less virulent, and the prognosis is less anxious.

Malignant growths are most commonly found in the ethmoidal region (where, however, the sarcoma seems to be more frequent than the carcinoma); the maxillary antrum; the floor of the nose; and the septum, especially on the edge of a perforation. But, as a matter of fact, cancer may originate in any part of the nasal cavity, and it may also be found that the nasal cancer is merely an extension of a growth originating elsewhere, as in the nasopharynx.

Symptoms.—The symptoms are the same as those we have just described in the section on the locally malignant neoplasms of the nose, but in addition, glandular enlargement may accompany the growth in the nose; emaciation will be more rapid; and, above all, recurrence after radical removal is probable.

It often happens, as when the growth comes under our notice early in its course, that the diagnosis must depend entirely upon the microscope, although the situation of the tumour, and its duration and speed of growth may give some information. Thus, a rapidly growing neoplasm of the antrum is usually highly malignant, and the chances are correspondingly poor, all the more so if the growth has attacked the bone and infiltrated the soft tissues of the cheek, or if it has found its way into the cavity of the nose or into the orbit.

Diagnosis.—Simple mucous polypi may appear in consequence of a malignant growth hidden away somewhere in the background, in which case the presence of the latter may never be suspected until more obvious signs arise. If mucous polypi are associated with frequent epistaxis or with a sanious offensive discharge, the possibility of malignant disease ought to occur to us. But when sarcomatous or carcinomatous masses themselves assume a polypoid form, they are easily distinguished from the simple mucous polypi by being fleshy, very vascular, and tender on probing.

Recurrent epistaxis from malignant disease may be mistakenly attributed to simple septal hæmorrhage or to some general disease. For that reason, it may be laid down as a clinical rule of practice: **when there is epistaxis, do not rest satisfied until you have actually located the bleeding spot.**

A syphilitic gumma responds rapidly to treatment, and shows no tendency to exuberant overgrowth as do the cancerous neoplasms.

Treatment.—Radical removal of the growth and its extensions is necessary, whether it is malignant or only semi-malignant. This is effected by one or other of the following operations, according to its situation and size.

Small growths, such as epitheliomatous ulcers on the septum, or on the inferior turbinal, may be radically removed by intranasal operation, the tumour, along with a sufficiency of the healthy tissue around it, being eradicated in one piece.

In the majority of cases, however, satisfactory access to the neoplasm can only be obtained by one of the following external operations.

LATERAL RHINOTOMY (MOURE'S OPERATION)

Description.—The exposure of the nasal cavity through the side of the bridge of the nose.

Indications.—Malignant or other tumours of the ethmoidal and sphenoidal sinus regions, and of the naso-pharynx.

Operation.—The patient lies on the back with head and shoulders a little raised. The surgeon stands on the side to be operated on. The anæsthetist sits opposite to the surgeon; the anæsthetic (chloroform) is administered at first on a mask, later through a Junker apparatus. The assistant stands at the head of the table.

Preparations must be made to cope with free hæmorrhage. To this end at least a dozen marine throat-sponges on George holders are got ready and placed in charge of a nurse, whose sole duty is to attend to them. The skin of the face is prepared by iodine-spirit solution.

If the growth is in the ethmoidal region, the posterior naris is blocked by means of a strip of gauze inserted through the nose. This, if practicable, is less likely to embarrass respiration than a captive sponge in the naso-pharynx. A tampon soaked in adrenalin solution (1-1000) is inserted into the ethmoidal region, and is placed against the inner surface of the nasal bone.

The anterior naris of the side operated on may be packed during operation with gauze, to prevent the blood running down and inconveniencing the anæsthetist.

With these arrangements, the whole of the blood will emerge at the operation-opening in the side of the nose.

Having surrounded the operation-field with sterile cloths, disposed so as to allow the anæsthetist access to the mouth and one eye, an incision is made as figured in Fig. 95; that is to say, from the inner end of the eyebrow, curving round the inner margin of the orbit, and thence straight down along the junction of the side of the nose with the cheek to the ala of the nose, curving round the junction of the ala with cheek and lip, and so into the floor of the vestibule. A second incision is sometimes made proceeding from the first incision at the level of the inner canthus, and curving along the lower margin of the orbit to the junction of the middle and outer third of that margin. This second incision is, however, unnecessary unless the growth extends below the level of the middle turbinal. Indeed, in most cases, a simple and straight vertical incision will be found to be sufficiently long without the curve round the ala. (Fig. 96.)

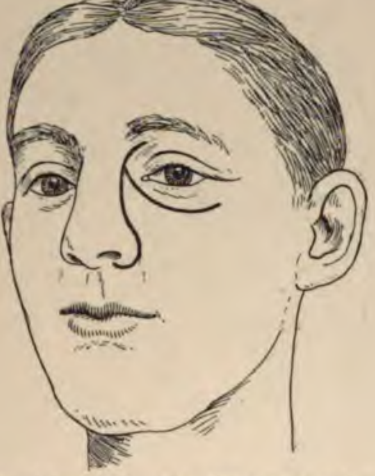


FIG. 95.—Moure's Incision for lateral rhinotomy.

In any case, the skin incision is rapidly deepened until the bone is exposed, and the angular artery and vein and several other branches of the facial vessels which are divided are clamped.

The flap marked out by the incisions is reflected by means of an elevator, and the soft tissues on the side of the bridge of the nose are similarly raised up to the middle line, both being held aside by retractors. The nasal bone and the ascending process of the superior maxilla are now completely exposed.

By means of bone-cutting forceps, inserted under the lower edge of the nasal bone at the pyriform aperture, the bones are rapidly nibbled away, exposing the lining membrane of the nasal cavity. As much bone is removed as is necessary to give free access to the nose, but care is taken to avoid the lacrymal bone and sac and the infraorbital nerve and vessels, while the bone of the dorsum of the nose is also left in order to maintain the bridge.

Incision of the lining membrane of the nose from without by knife or scissors lays the interior of the ethmoidal region open to inspection, so that the situation of the growth in front can be clearly made out.

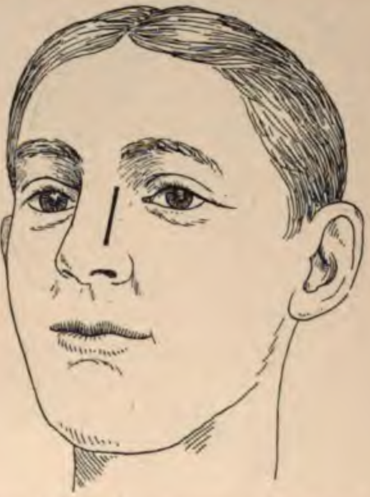


FIG. 96.—Lateral Rhinotomy. The simple incision.

Keeping between the lining of the nose and its bony wall, if that bony wall is still uneroded, the tumour is undermined and separated, in one mass if possible, from the lamina papyracea of the orbit and the cribriform plate above.

Finally, by means of a long, sharp spoon, its connections posteriorly are detached, and the tumour mass is removed through the opening in the side of the nose.

This process of separation and removal is attended with very free hæmorrhage, but, as in all nasal operations, the surgeon does not pause to arrest it; he hastens on to the completion of his task, knowing that the bleeding will not cease until all the pathological tissue has been removed.

The final step is to wipe out and to inspect the interior of the nose in order to make certain that all the tumour growth has been removed. Special regard must be paid to possible extensions of the growth into the orbit, into the cranial cavity, or into the antrum or sphenoidal sinus, such extensions, if found, being followed up and removed by blunt elevation and separation. It sometimes happens that what at first sight may seem to be an extension of the growth, proves to be in reality the original starting-place and main mass of the tumour, necessitating a much wider removal than had been anticipated, with a fresh renewal of the hæmorrhage.

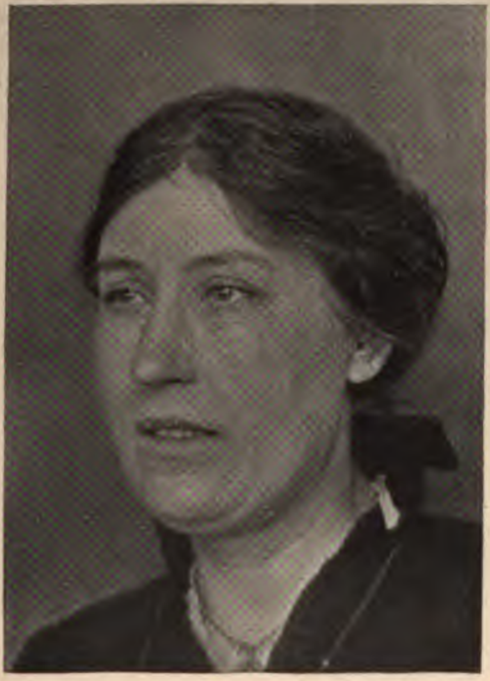


FIG. 97.—Scarcely perceptible scar of lateral rhinotomy. Operation was performed for ethmoidal epithelioma—Untouched photograph.

The growth and its extensions having thus been thoroughly removed, the skin flaps and incisions are brought accurately into apposition, and sutured with fine silkworm gut or horsehair.

A collodion dressing is sufficient, as healing of the face wound takes place with rapidity. The scar is inconspicuous, and the gap in the bone does not lead to any depression of the surface. (Fig. 97.)

The gauze strips are removed from the nose as soon as the operation is finished, and are not re-inserted unless there is a continuation or recurrence of hæmorrhage.

Results.—The immediate results are excellent; the remote results depend upon the nature of the growth and the thoroughness of its removal.

The patient is kept under observation, and if a recurrence is detected, it may be removed with a curette under nitrous oxide or even local anæsthesia. In the less malignant forms, the methodical destruction of such colonies as these which had escaped the first operation will finally lead to cure.

In the more malignant growths, if recurrence takes place, it does so both locally and in the lymphatic glands of the neck, and leads finally to death.

ROUGE'S OPERATION

Description.—The reflection upwards of the upper lip and point of the nose to give access to the lower parts of the nasal cavity through an incision beneath the upper lip.

Indications.—Rouge's operation is used for extensive growths about the floor and inferior meatus of the nose.

Operation.—Preliminaries as for the radical antrum operation. (See p. 334.)

The upper lip being turned up, an incision is made along the gingivo-labial recess, having its middle at the frenum of the upper lip, and extending as far as the level of the first bicuspid tooth on either side. The incision is carried down to the bone, and by means of an elevator, the soft tissues are separated from the osseous margin of the pyriform aperture.

The lining of the nose is cut around the aperture to give access to the inferior meatus, and with scissors or knife the cartilaginous septum is cut through from the anterior nasal spine below to just under the bridge above. This, with a little further clearance laterally permits of the upper lip and the point of the nose being detached in a flap upwards, where it can be held by an assistant while the nose is being cleared.

A sponge in the naso-pharynx may be required to prevent blood finding its way into the pharynx, but in this operation also it may be possible to block both posterior nares with gauze.

The nose, having been cleared of new growth, the point of the nose and the lip are replaced, the edges of the wound being secured by sutures. The septum wound does not require stitching.

REMOVAL OF THE SUPERIOR MAXILLA

may be required when the maxillary antrum is the seat of epithelioma.

Position.—The patient lies on his back with head and shoulders raised.

The *Anæsthetic* (chloroform) may be administered through a Junker tube; through a laryngotomy opening; or by the intra-tracheal method.

One or two surgeon-assistants are required. The operator stands on the patient's right hand.

Operation.—The upper lip is first of all completely divided in the middle line from the columella to the free edge of the prolabium. While the surgeon is so doing an assistant compresses the lip and with it the coronary arteries on either side of the middle line. After the lip is divided, the arteries are at once caught.

From the upper end of the lip incision, an incision passes along the lip across the floor of the nasal vestibule outward to the edge of the ala, and thence round the ala, and up alongside the nose to a point half an-inch below the inner canthus of the eye. From the upper end of this incision, a second incision is made along the lower margin of the orbit to near the outer canthus.

These incisions are rapidly deepened to the bone, the vessels met with being secured by forceps; they include the angular artery and vein, the lateralis nasi artery and some smaller branches. Bleeding is lively for a few moments, but the points are easily seen and caught. The soft tissues of the cheek are raised from the bone by dissection.

The maxilla is now to be separated from its bony attachments.

First, the edge of the pyriform opening is cleared with the knife, and the nasal process is cut through with a fine saw, between pyriform opening and orbit.

The lower edge of the orbit is next defined, cleared, and the periosteal floor of the orbit raised, and the bony floor is cut through from the point where the nasal process has been divided to the spheno-maxillary fissure by means of a fine chisel. Next, the malar bone is divided with a saw from the surface to the spheno-maxillary fissure, the division running obliquely downwards and outwards.

The palatal attachments remain. For their severance, the central incisor tooth on the diseased side is removed, and the mucous membrane and periosteum of the hard palate is divided in the middle line from the incision in the lip as far back as the soft palate.

The soft palate is left behind, and for its separation, a transverse incision, starting from the posterior end of the median palatal incision, is carried right through the soft palate.

(Sometimes it is possible to save the muco-periosteal roof of the mouth.)

An incision made with a long-bladed knife is carried through the muco-periosteal floor of the nose throughout its whole antero-posterior length, from the incision in the lip. Through this nasal incision a fine saw effects from above downwards the division of the hard palate.

Finally, with lion forceps holding the bone by the alveolus below and the orbital palate above, the bone is wrenched out and freed of its other bony connections. Some soft tissue attachments at the back usually also require division with scissors. Bleeding points in the cavity are caught and ligatured if possible, but what bleeding there is usually stops after firm packing with iodoform gauze.

The skin incision is finally closed, the cheek-flap covering the gap in the facial skeleton.

After-Treatment.—The packing is removed in twenty-four hours, and not again inserted unless required for hæmorrhage. The cavity may be douched out daily from the mouth.

The *Results* depend upon the nature and extent of the growth. If confined to the antrum, non-recurrence is occasionally secured. But in most cases the disease recurs locally.

So far as the operation itself is concerned, rapid recovery is the rule.

NASAL NEUROSES

Anosmia.—Loss of the sense of olfaction may be (1) *obstructive* or (2) *perceptive* in origin.

1. Any cause hindering or preventing the access of odoriferous particles to the olfactory region of the nose—which occupies

quite a small area in the roof, septum, and upper ethmoidal region—such as hypertrophic rhinitis, hypertrophy of the middle turbinals, polypi, and other swellings and tumours, will induce hyposmia or anosmia, and the treatment consists in the removal of the obstruction.

2. Of the perceptive variety, the causes may be (a) *local*, as in atrophic and other forms of rhinitis, which injure the end-organ of the olfactory nerves; (b) *neuritic*, as after influenza; (c) *central*, from hysteria, tabes dorsales, general paralysis of the insane, basal meningitis, cerebral tumours, etc.

The treatment of this variety is unsatisfactory, and save when it is due to local inflammatory causes, the return of the sense should not be expected.

Hyperosmia, or hypersensitiveness of olfaction, is sometimes complained of in hysterical affections.

Parosmia—perverted sense of smell—occurs in some forms of insanity.

Cacosmia—the subjective sensation of fœtor—is not infrequently experienced by sufferers from accessory sinus suppuration. Objective fœtor—that is, fœtor which is imperceptible to the patient, although every one else may be aware of it—is one of the characteristics of ozænatous rhinitis. (See p. 282.)

Rhinorrhœa is the name given to the flow of watery fluid from the nose. Three varieties may be distinguished.

1. Chronic hypertrophic rhinitis may be attended with free secretion of a clear watery fluid soaking many handkerchiefs in the course of the day. The treatment consists in the removal of the cause.

2. VASO-MOTOR RHINORRHŒA (VASO-MOTOR RHINITIS)

In this form the fluid is serous, and is poured out from the middle turbinal and the upper regions of the nose. (As a matter of fact, thin, watery nasal secretions always do proceed from the ethmoidal region.)

The condition is common, its cause unknown, and its cure uncertain. Sometimes it seems to be due to the irritation of a septal spur or deflection. Sometimes it is obviously the effect of some autotoxin, as when it is associated with attacks of asthma. The similarity its symptoms then present to those of hay fever, and to the phenomena of anaphylaxis, give support to the autotoxic theory.

Symptoms.—At a certain period of the day, usually after getting up in the morning, the patient is seized with an intense irritation in the nose, which sets up violent and prolonged sneezing attended with a free flow of watery fluid from both nostrils, sometimes with irritation and lachrymation in the eyes, and occasionally also with wheezing in the chest, with coughing, and even with definite asthmatic breathing.

The paroxysm lasts for about half-an-hour or an hour, gradually subsiding and disappearing until next day about the same time, although in some cases the tendency to recurrence persists all day, and a fresh attack is set agoing by trivial causes.

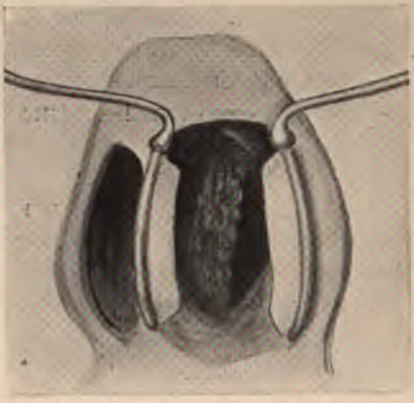


FIG. 98.—Granular œdema of the septum in long-standing paroxysmal rhinorrhœa.

On examination, the nose, during the attack, is seen to have the turbinals pale, watery and swollen.

The disease presents the same phenomena as hay-fever, but it differs from hay-fever in continuing all the year round, and in being as severe in winter as in summer. It lasts for many years, and it is liable to lead to serious secondary results, as multiple polypi frequently form, and the nasal mucosa undergoes a typical and peculiar change of what may be termed "granular œdema." (See Fig. 98.) This condition still further aggravates the severity of the attacks.

Diagnosis.—We have already indicated the distinction between hay-fever and paroxysmal rhinorrhœa, and the characteristic sequence of events as we have described them renders diagnosis easy.

Prognosis should be guarded. Sometimes local operative treatment cures; or the attacks may be modified by attention to general health, or by drug treatment. Sometimes the attacks defy all remedies.

Treatment.—If the septum shows a deflection or spur, and if the turbinals are enlarged or polypoid, local treatment directed to the removal of these abnormalities may be followed by great relief and even by cure. In the condition of granular œdema it is advisable also to perform submucous resection of the septum, even if it is not deviated, as experience shows that the operation may effect considerable improvement in the attacks.

Alexander Francis recommends light cauterization of the septum for this disease, as he does for asthma. (See also p. 307.)

If local measures fail, or seem to be unnecessary, benefit may be obtained from belladonna or atropin internally, from potass. iodid. in small doses, or from calcium chloride or lactate.

3. In **Cerebro-spinal Rhinorrhœa**, a rare condition, the fluid discharged is cerebro-spinal fluid. In this form the rhinorrhœa is generally unilateral, and the flow is almost continuous for weeks, months, or years. Headache and symptoms referable to the central nervous system may be present (Sir St. Clair Thompson). The fluid, when collected and tested, shows the character and gives the reactions of cerebro-spinal fluid. These are: a low specific gravity (1003 to 1004), the absence of albumen (or if albumen is present, there is no more than a trace), and the property of reducing the copper in Fehling's Solution. (See also p. 558.)

HAY FEVER (HAY ASTHMA)

Although generally numbered among the nasal neuroses, hay fever is a specific disease due to a substance in the pollen of certain grasses and other plants which has a toxic and irritating effect on the nasal, conjunctival, and bronchial mucous membranes of those susceptible.

In addition to pollen, the volatile exhalations of some animals exercise an irritating influence upon the exposed mucous surfaces of certain people. Thus, an asthma due to horses is recognized, and cats also are said to induce it. In such cases, the attacks appear only when the patient comes near the animal in question, and it is important to note that sufferers from horse asthma are peculiarly susceptible to the influence of horse serum, and are made seriously ill with anaphylactic symptoms if they receive diphtheria antitoxin, or other antitoxins made of horse-serum.

NASAL ASTHMA

A certain proportion of sufferers from asthma manifest nasal abnormalities, such as spurs, deviations of the septum, polypi, or such conditions as paroxysmal rhinorrhœa or hay fever.

In a few of these cases, the cure of the nasal disease results in the cure of the asthma. In a larger number the removal of the nasal abnormality is followed not by cure but by a moderation in the severity of the asthma, so that although the attacks do not cease, they are more easy to endure. In a few cases, the cure of the nasal disease has no effect whatever upon the asthma, and cases have even been recorded where the nasal treatment seemed to have made the asthma worse.

If the history shows that the asthma and the disease in the nose appeared at the same time, such as may be found not infrequently in a case of nasal polypi, the chances of curing the asthma are good, and those chances are all the better if the duration of both disorders has been brief.

Again, if an asthmatic patient is found to have a deviated septum or a septal spur, or an enlarged middle turbinal, especially if points are visible where two opposing surfaces are in contact, nasal operation may lead to considerable relief from the asthma, and sometimes even to complete cure. It is not justifiable to perform any surgical operation upon the nose in asthma unless some undoubted nasal abnormality or disease is present. But this injunction does not apply to

Cauterizing the Septum, as Alexander Francis has described it.

Method.—Under light cocaine anæsthesia, a fine galvano-cautery terminal is brought to a cherry-red heat under inspection within the nose. But it is not placed in contact with any surface until the electric current is shut off, and then just as the platinum terminal is ceasing to glow, it rapidly and lightly touches the septum at a spot approximately on a level with the anterior end of the middle turbinal. Hereabouts one quite commonly finds in asthmatic people an œdematous patch, but often no such indication of abnormality is visible.

Results.—If one is fortunate enough to hit the right spot, the effect of this gentle cauterizing upon the asthma is said to be surprisingly good. My results, personally, have not equalled those claimed on good evidence by Francis, but I have sometimes obtained a decided and prolonged mitigation in the severity of the attacks.

Francis lays stress upon the feather-touch of the cautery, and believes the improvement to be due to a reduction in blood-pressure reflexly brought about by the cauterization.

The Adrenalin Treatment of Asthma.—(a) *Intranasally.*—A freshly made solution of adrenalin chlor. in water (1-10,000 or stronger) may be sprayed into the nose at the outset of an asthmatic paroxysm with benefit.

(b) *Hypodermically.*—Striking and immediate relief to the bronchial spasm follows the hypodermic injection of from five to eight minims of sol. adrenalin. chlor. (1-1000), and its repeated and continued use does not seem, so far as recent reports go, to be productive of any ill effects, except a severe headache when too large a dose is employed. It is therefore preferable in every way to cocaine or morphine.

In operations under general anæsthesia upon asthmatic patients, a paroxysm usually sets in as the patient is recovering from the anæsthetic, and alarming cyanosis may develop. A timely hypodermic of adrenalin removes the dyspnœa as if by magic.

Nasal Cough is allied to nasal asthma in being due to irritation of some part of the interior of the nose. Polypi may be the exciting cause, or a turbinal enlargement or a septal spur or deviation. It is sometimes possible to evoke the cough, and so to assure oneself of the diagnosis, by touching certain spots in the nose. Cocaine naturally is not employed, and the agent used is a cotton-wool covered probe. The touch must be gentle.

The spots are: the anterior end of the inferior turbinal; the posterior end; the corresponding area on the septum; the anterior end of the middle turbinal and the opposite area on the septum (the *tuberculum septi*—the same region as is cauterized by Francis for asthma); and finally the orifice of the Eustachian tube. If touching any of these spots sets up coughing, lachrymation, violent sneezing or other signs of excessive reflex reaction, nasal hyperæsthesia may be diagnosed, and except in the last, may be treated by local applications of the cautery or by removal of the irritable spot.

Nasal Headache.—Headache and neuralgic pains in any of the areas supplied by the fifth nerve may radiate from a point of irritation or pressure within the nose (L. H. Pegler). Consequently, in cases of causeless headache, faceache, and neuralgia, any apparent centre of irritation within the nose, such as spurs exerting pressure ; deflected septa ; and what not, should be removed or rectified. (For headache due to disease of the nasal accessory sinuses, see p. 314.)

Synechiæ of the Nose are adhesions which have formed across the nasal passage between septum and turbinals or lateral wall. They are usually due to adhesion of two opposing surfaces denuded by operative measures or by disease, but sometimes no cause can be assigned for their presence. They vary from thin friable threads to thick obstructing bands of dense fibrous tissue.

Symptoms.—Sometimes synechiæ induce no symptoms. At other times they seem to cause an amount of obstruction out of all proportion to their size. If extensive, they naturally produce considerable nasal obstruction.

Their presence may interfere seriously with the examination and treatment of a nasal disease.

Treatment.—Sometimes simple division suffices to obliterate them permanently, but more often they will re-unite as fast as they are divided unless some object be interposed between the raw surfaces. For this there is nothing better than a piece of india-rubber from the palm of a surgical rubber glove, worn continually for a week or ten days, being taken out, cleansed, boiled, and replaced daily between the raw surfaces.

The adhesions may be divided with the knife, the galvano-cautery, or the diathermy terminal.

INTRANASAL DACRYOCYSTOTOMY

is performed for stenosis of the nasal duct. It consists in making a permanent opening between the lacrymal sac and the interior of the nose in front of the middle turbinal. It is indicated in all forms of epiphora due to obstruction of the duct, whether the duct and sac have become infected or not.

The operation should be performed before slitting the canaliculus, and in preference to removal of the sac.

Operation.—The patient lies on his back with head and shoulders a little raised ; the surgeon stands on the right hand side, or, if he prefers, on the side to be operated on.

If a general anæsthetic (chloroform) be employed, the anæsthetist is on the opposite side to the surgeon and a Junker apparatus is employed. Local anæsthesia suffices, however, and it is necessary even when a general anæsthetic is employed.

The site of the lacrymal sac corresponds to an area on the outer wall of the nose immediately anterior to the middle turbinal. (Fig. 99.) Here the sac may be in direct relationship with the bony wall of the nose and the nasal mucosa, but quite frequently an ethmoidal cell is interposed, and has to be traversed by the operator.

Local anæsthesia is induced by the application of cocaine-adrenalin paste to this area together with the injection of a few drops of 10 per cent. sol. cocaine with an equal quantity of sol. adrenalin (1-1000) by means of a lacrymal syringe into the sac through the canaliculus.

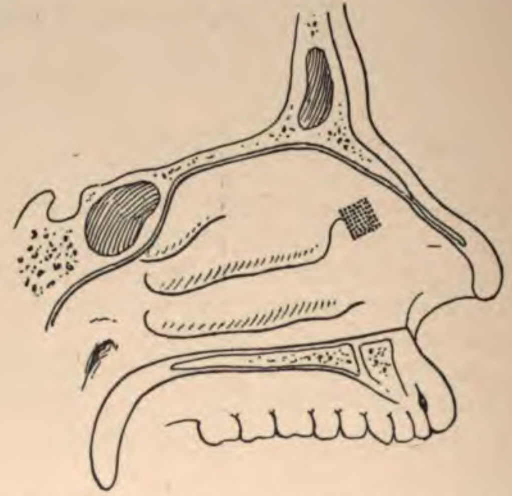


Fig. 99.—Nasal Dacryocystotomy. Approximate site of lacrymal sac and operation indicated by shading.

With a sharp knife passed into the nose under inspection a small flap of mucous membrane with the base below, is turned down from the area referred to. With a long recurved chisel and a mallet, the underlying bone thus exposed is removed so as to form a window. If there is no ethmoidal cell interposing, this lays bare the inner wall of the lacrymal sac.

A lacrymal probe is then passed through the inferior canaliculus into the sac, and pressing on the inner wall makes it project into the nose through the window in the bone we have just made.

The projecting sac wall being thus made visible, it is seized from within the nose with a special pair of sharp-toothed catch-forceps, and secured, while a satisfactory piece of sac wall is cut out either by fine-bladed scissors, or by a fine bistoury such as that used for submucous resection of the nasal septum. The flap of the mucous membrane may then be replaced, and retained with a light packing.

The object of the flap is to prevent granulation-tissue formation from the cut edge of the bone as that readily leads by cicatrization to the closure of the artificial opening in the sac. But as a matter of fact granulation-tissue always does form, and the best method of preventing cicatricial closure is to inspect the nose once a week for two or three months after the operation, and under cocaine to pick away the granulation-tissue as it forms, until finally the wound heals (J. B. Horgan).

Difficulties.—As an operative procedure dacryocystotomy presents the most extraordinary variations. Some cases are easily accomplished, while others are most tedious and difficult.

An ethmoidal cell may be interposed, and then when it has been opened up, the lacrymal sac lies at the bottom of a small fossa, the side of which prevents our view of the sac. This side must then be chiselled down until the sac wall is exposed.

Sometimes a fossa of this kind exists without any ethmoidal cell, simply through the prominence of the ascending process of the superior maxilla, the bone of which is very hard and difficult to pare down with chisel or gouge.

In the most difficult cases, the operator may find it easier to abandon operation under inspection and to operate under the guidance of the finger placed externally over the lacrymal groove. By exercising deep pressure, it is possible indeed to perform the whole operation with but occasional inspection of the interior.

Granulation-tissue may go on forming interminably after the operation, in spite of weekly efforts to reduce it. In that case, we may bring about its cessation by removing a piece of the anterior end of the middle turbinal.

Results.—In most cases epiphora is cured by the operation. In some, however, it continues, or after a few months' reprieve, begins again. In such cases, the operation should be done again; as the recurrence is probably due to the cystostomy opening having contracted.

Epiphora in Intra-nasal Disease.—Epiphora is occasionally found in intra-nasal disease, especially in the following: ethmoiditis; syphilis; lupus; neoplasms such as fibroma, carcinoma, sarcoma, and after operations on the maxillary antrum.

OPERATIONS ON THE PITUITARY GLAND

It is possible to reach the sella turcica through the nose, and this is the route usually chosen nowadays in operating on cysts or tumours of the pituitary body.

A tumour of the pituitary body induces the symptoms of brain tumour with early eye symptoms, together with the phenomena of profound constitutional disturbance such as acromegaly, gigantism, adiposity, and so on, from the withdrawal or perversion of the pituitary secretion. These phenomena leading to an X-ray examination of the skull, distension of the sella turcica and other changes may be discovered.

The operation consists in approaching the sella turcica by means of a submucous resection of the nasal septum (Fig. 83), carried back to, and through, the sphenoidal sinuses, the septum of which is removed to expose freely their posterior wall. The breaking down of this wall lays bare the sella turcica, where cysts, if present, may be opened, and solid tumours reduced by curetting, with much relief to the symptoms, and even in some few cases with cure.

The operation may be performed in stages, at intervals of two or three weeks, each stage being effected under local anæsthesia.

The chief difficulty consists in opening the sella in or near the middle line, so as to avoid the cavernous sinus. This step may be facilitated if performed under the guidance of X-rays.

More room for operating can be secured by removing turbinals and ethmoidal cells a few weeks before the submucous resection is performed.

The chief danger is post-operative meningitis, as it is difficult to keep this deep wound aseptic.

FOREIGN BODIES IN THE NOSE AND RHINOLITH

Children are fond of inserting foreign bodies into the nostrils. If noticed at the time, inspection will, of course, reveal the situation and nature of the body; but if, as is not uncommonly the case, no one has observed the circumstance, the foreign body is allowed to remain, and after some years it becomes incrusted with lime salts, forming what is known as a *rhinolith*. But although the nucleus of a rhinolith is almost always a foreign body, this is not invariably the case. Rhinoliths may be removed which on section show no signs of any nucleus.

Symptoms.—Both foreign bodies and rhinoliths cause a unilateral muco-purulent discharge, with more or less nasal obstruction, and possibly some pain or occasional epistaxis. On examination, the presence of the foreign body may be obscured by copious discharge, or by swelling of the turbinals; but the cleansing of the nostril, the application of cocaine to induce a shrinking of the swollen turbinals, and the cautious use of the probe, will generally result in the discovery of the body. As a rule, it will be found in the inferior meatus, fairly well back in the nose.

Removal is always painful and sometimes difficult, but it can usually be accomplished with a bent director inserted beyond the body. If, however, the substance is impacted, or if it is too large to be readily hooked out, a general anæsthetic should be given, and the mass extracted deliberately, being broken up with strong forceps if necessary and removed piecemeal. Every effort should be made to avoid increasing the damage already inflicted on the nasal mucosa and turbinals by the foreign body, as adhesions readily form, and at a later date may seriously obstruct the nasal passage.

Maggots in the Nose.—In warm climates, certain species of blow-fly are readily attracted to the nose, when, as in the ozæna, the organ is disseminating an odour of putridity. The eggs laid in the nostril hatch out larvæ, which set about devouring the lining tissues of the interior of the nose, and if not got rid of, they cause great destruction, which may even involve the meninges and lead to death.

The presence of the insects is unsuspected until the symptoms set in. These consist, first of all, of intolerable itching with nasal discharge, at first watery, and later purulent and blood-stained. Examination of the nose may reveal the living maggots, but in any case the destruction they cause and the consequent pain and distress they induce sooner or later lead to their discovery.

Treatment.—The patient's head should be inverted, and his nasal cavities filled with oil, as this kills the larvæ, and they can then be removed by syringing. Unless they are killed, syringing will fail to dislodge them.

CHAPTER IX

EXAMINATION AND DISEASES OF THE NASAL ACCESSORY SINUSES

Causes of Sinusitis.—The close proximity of these cavities to the nose, which is so liable to catarrhal inflammation, and their anatomical formation predispose to the occurrence and favour the persistence of catarrh of their lining membrane, and this readily becomes transformed into purulent inflammation.

Acute catarrhal rhinitis is frequently accompanied with, and followed by, an extension of the disease to one or more of the accessory sinuses and the consequent stenosis of their ostia, with retention of the mucous secretion. A chance infection with one or other of the pyogenic organisms will then readily lead to a suppurative inflammation of their lining, and this, if not speedily relieved, naturally or artificially, will induce all the long-drawn-out and troublesome phenomena of chronic sinus suppuration.

Purulent sinus disease may also follow the acute infectious fevers. In the case of the antrum, carious teeth of the upper jaw may set up suppurative disease by direct infection from the root of the decayed tooth. Finally, sinuses may be infected from their neighbours, the process spreading from one to another by direct extension along the mucous membrane lining their ducts; through their lymphatic connexions; or by direct passage through a bony wall from one cell to another.

Pathology of Nasal Sinus Suppuration.—Our knowledge of the pathogenesis of sinus suppuration is scanty. We do not know why one patient with nasal catarrh develops the disease while another with nasal catarrh equally severe or persistent does not, and bacteriology does not help, since all that it informs us is that the organisms found are the usual pyogenic bacteria.

Moreover, it is the fact that when one sinus is affected, the disease frequently shows a tendency to pass, as we have just said, from that one to another. But this also is by no means invariable. The maxillary antrum, or the frontal sinus of one side, for example, is often the only sinus affected, and although from it pus may have been emerging in close contact with the ethmoidal cells, perhaps for months, yet these cells may remain uninfected.

When the ethmoidal cells do become infected, however, the tendency is for the infection to spread, upward into the frontal, backward to the sphenoidal, and downward into the antrum, if that cavity is not already diseased.

Further, the ethmoidal cells are undoubtedly more frequently affected, and so, presumably, are more vulnerable, than any of the other sinuses.

Another notable point in the natural history of nasal sinus suppuration is that the process is essentially a suppuration of the soft parts of the mucous lining. It is true that the bone shows microscopic evidences of disease in round-cell infiltration, and, at times, rarefying osteitis. But these osseous changes are not the prominent feature in the pathological picture; they are, therefore, quite obviously subsidiary and secondary, not only in time, but also in importance, to the changes in the mucous lining. And this seems all the more remarkable when we recall that the bone of the walls of many of the sinuses is thin and papyraceous, and therefore, it might be supposed, very liable to cario-necrotic processes when the cells become infected.

Nevertheless, the bone may, and does become at times, seriously and even gravely infected as we shall see, osteomyelitis being induced, either localized or spreading, but osteomyelitis as a consequence of sinus suppuration, is, in reality, quite rare. The great majority of cases show no such tendency. Sinus suppuration, in other words, remains a mucous membrane suppuration throughout.

In the mucous membrane, however, the pathological changes induced are profound and destructive. The thin, fine lining of the cells becomes swollen and turgid. Without losing its epithelial covering, it becomes hypertrophied and œdematous until by the combined action of inflammatory œdema and of gravity, whereby a passive œdema also is super-induced, it undergoes a relatively enormous overgrowth, and appears to our eye as a group of mucous polypi.

This process taking place simultaneously throughout the ethmoidal system, for example, will induce a widespread and rapid growth of these polypi until, it may be, the whole of the ethmoidal region is occupied by vegetations the increasing bulk of which destroys, by pressure, the normal anatomical arrangement of the ethmoidal labyrinth, and may even expand the nasal bones externally until the bridge of the nose itself becomes broadened and deformed.

In such cases, when the affected region is being cleared by the surgeon of its polypi, spicules and pieces of disintegrated bone will be removed in and among the polypi and polypoid mucous membrane. But such bone is still alive. It is not necrosed, albeit thinned and broken up by the pressure, partly of the round-cell infiltration, partly of the polypoid expansion.

Thus, we are justified in supposing that the local resistance to the infection must be very strong, and that the persistence of the disease is probably due more to the anatomical peculiarities of the regions involved than to any lack of resistance to pyogenic organisms.

The local peculiarities consist in the number and complicated relationship and arrangement of these cells and sinuses; of the fact that in some of them, such as the antrum and the sphenoidal sinus, the natural orifice of the cavity is situated too high for efficient drainage (Figs. 102 and 123), while in all of them the orifices are so small that a little swelling of the lining is sufficient to close them entirely. Finally the shallow depth of the mucous membrane of the sinuses, and its close attachment to underlying bone, furnishes little or no space for the accommodation of inflammatory products. And thus when infection of the mucous lining does take place, drainage is readily and rapidly inhibited, and the infection is locked in, partly as a consequence of its own processes and partly because of the anatomical disposition of these mucous-lined cavities.

Acute Sinusitis usually owes its presence to infection during the course of acute coryza (catarrhal fever). The sinus disease may either be a first appearance or an acute exacerbation of a chronic sinusitis.

The organisms found, sometimes in pure culture, are the pneumococcus, streptococci, and, in influenza epidemics, Pfeiffer's bacillus.

The attack varies considerably in severity in different cases, sometimes being attended with severe pain, prostration, and high and prolonged fever, at other times producing only some dull aching and local tenderness. The worst cases are those in which the disease attacks the bony wall of the sinus cavity, producing osteomyelitis, softening, caries, and the extension of the infection beyond the limits of the sinus, the so-called *sinusitis exulcerans*. This development, which is infrequent, is most commonly seen in acute inflammation of the frontal sinus. (See later.)

On rhinoscopy, one may find signs of acute purulent rhinitis. There may be œdema and swelling of the middle turbinal, and these signs, especially if unilateral, should always arouse suspicion of sinus infection. But sometimes no signs are present at all in the nose.

Diagnosis.—The common error is to regard the pain of acute sinusitis as simple neuralgia, especially in acute frontal sinusitis.

The *prognosis* in acute sinusitis is in general favourable, as most cases get well spontaneously; but a few pass into chronic suppuration; while still fewer lead on to serious complications and death, if unrelieved.

Treatment.—Heat locally often relieves pain, and the application of a tampon soaked in cocaine, 10 per cent. solution, with an equal quantity of adrenalin solution (1-1000), or of the cocaine-adrenalin paste (see p. 226) to the neighbourhood of the nasal orifice of the sinus, by shrivelling-up the swollen lining of the ostium, may permit of an escape of pus from the affected sinus.

If such simple remedies fail, and the pain continues, the affected sinus or sinuses should be opened up for drainage by the intranasal route as detailed below. To this general rule of operative treatment of acute sinusitis by intranasal operation only, there is one important exception, namely, in *acute frontal sinusitis* with evidence of extension beyond the limits of the sinus to the soft parts of the orbit, forehead, or brain. (See below.)

Chronic Sinusitis.—In chronic purulent sinusitis the bacterial infection is mixed, and the disease tends, if left untreated, to persist indefinitely. Oedematous swelling of the mucous lining of the sinus and of its orifice or duct, passing on to polypus formation, is usual, while the secondary effects of toxic absorption, such as headache, mental dullness, general ill-health, orbito-ocular disease, and middle ear disease, chronic pharyngitis, laryngitis and bronchitis, and even rheumatism, are frequent complications. One or other, or a combination, of such secondary, remote phenomena may be, in fact, the only symptom for the relief of which the patient consults his medical adviser, so that the real source and origin of the illness may remain undiscovered unless particular attention be paid to the nose, and the patient be questioned as to the presence of nasal discharge or nasal obstruction.

But while it is true that many cases of sinus suppuration are obscure and difficult of diagnosis, it is also true that in many others the signs and symptoms, both local and general, are so plain and unmistakable that the real nature of the disease is soon evident Suspicion of sinus suppuration, then, having been aroused, we proceed to make sure, first, whether there is actually sinus disease present; secondly, which group of sinuses it is that is affected; and thirdly which member or members of the group have been selected by the disease.

With regard to the first point, it may be stated that a chronic nasal discharge of pus or muco-pus always means either sinus suppuration; or caries or necrosis of bone, usually syphilitic; or a foreign body. If the others can be excluded, we may be certain that the pus is proceeding from one or more of the accessory sinuses.

In this connexion however, it is necessary to remark that purulent sinusitis may exist, and the patient may complain of purulent discharge from the nose, and yet when we come to examine the nose, no pus or any other abnormality of any kind can be discovered. In such cases the patient should be asked to keep for the surgeon's inspection the nasal discharge he blows down, and if it is seen to consist of muco-pus, the presence of sinus suppuration may be taken for granted, and a systematic search made to discover which sinus or sinuses are involved, according to the directions detailed below.

X-Ray Examination.—In the investigation of a case of suspected nasal sinus suppuration, useful information can be obtained by means of the X-rays. Liquid, such as pus, and perhaps polypi, in the antrum or frontal sinus, throw a faint shadow, visible on the developed plate as a dullness of these cavities which renders their outlines blurred and indistinct as compared with the normal. (Figs. 101, 115.) Skiagraphy of the sinuses demands special skill and experience, for which reason the rhinologist ought himself to acquire the art of reading the skiagram. It is most useful in the diagnosis of antrum and frontal sinus suppuration, and in the latter it is of special value in revealing the extent and configuration of the sinus cavity.

The X-rays are of little or no value, however, in the diagnosis of posterior ethmoidal and sphenoidal sinus suppuration, the latter being always translucent whether it is the seat of suppuration or not.

Prognosis.—As we have already seen, chronic nasal sinus suppuration tends, if untreated, to persist indefinitely. Nay, more! It tends to pass from bad to worse, until, it may be, every sinus in the nose is suppurating (*pansinusitis*), and the entire cavity of the nasal chamber is blocked with numerous polypi, recurring as fast as they can be removed, while the patient, besides having to endure the miseries of complete nasal obstruction, is reduced to a low state of health as a result of the continual absorption of bacterial toxines.

When efficiently treated, however, by the opening up and draining of the affected cavities, the progress of the disease is arrested, until finally all suppuration ceases, although it certainly must be admitted that this welcome termination is nearly always very slow in coming

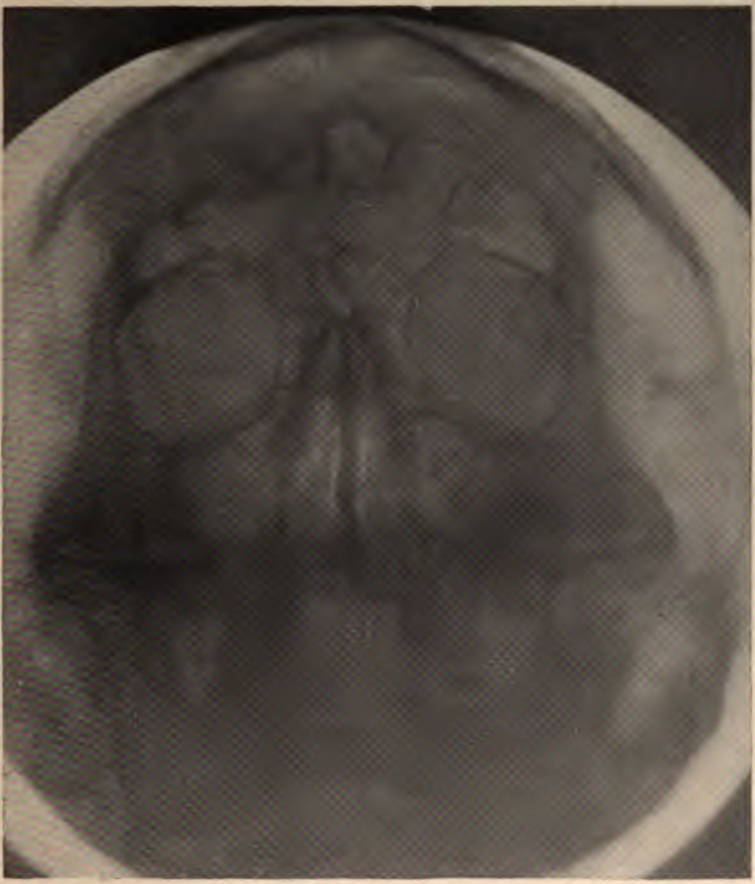


FIG. 100.—Skiagram (by Dr. Robt. Knox) showing normal maxillary antra and frontal sinuses; the latter are large and of irregular configuration.

The *Treatment*, generally speaking, of a suppurating sinus may perhaps best be summed up as drainage and ventilation. In this department of our work, however, the currents of expert opinion during the last twenty years have been very inconstant, Nor can it be said that they have settled into one stream even to-day.

Where it is possible to treat the disease by a total removal of the diseased sinus, as is the case with the small anterior ethmoidal cells, the result has amply justified expectation, and little or no difference of opinion and practice exists. But the same cannot be claimed with respect to sinuses like the maxillary antrum, the total ablation of which is impracticable. In this instance efforts have been made, and are still being made, with considerable but variable success, to eradicate the disease by a free opening up of the cavity from without in order to effect the complete removal of its diseased mucous lining.

A similar attempt in the case of the frontal sinus having been deemed unsatisfactory, however, an operation to obliterate that cavity was devised. But this radical experiment after many trials was found to be at once more dangerous to the patient than the simpler operation, and not any more successful in curing the suppuration. By most modern rhinologists it has therefore been abandoned, at all events for simple chronic suppuration of the frontal sinus.

Meanwhile, in dealing with the more deep-seated cavities such as the sphenoidal sinus and posterior ethmoidal cells, rhinologists found themselves confronted with sinuses that, on account of their anatomical relations, could neither be totally ablated nor have their diseased lining membrane removed. It was necessary, therefore, to be content with the simple opening up of these cavities in such a way as to ensure their permanent free drainage. Then it became evident that such simple measures were followed, in most cases, not only by a disappearance of the symptoms induced by pressure and toxic absorption, but even in the course of time, by the cure of the purulent disease itself. Consequently we have been led in recent years to try the effect of similar partial and conservative measures on the antrum likewise, and on the frontal sinus.

This brings us to the point of considering what are the principles that should guide us in carrying out the surgical treatment of chronic nasal sinus suppuration.

The first principle, that of permanent drainage with free aëration of the suppurating cavity, is admitted by every one.

But the second, which postulates as a necessity the meticulous removal of the infected mucosa of the suppurating sinus, is less unanimously accepted.

As we have seen, it can be dispensed with in the case of the sphenoidal sinus. Why then is it essential in the case of the maxillary antrum ?

Further, if the conclusion we reached in discussing the pathogenesis of nasal sinus suppuration be correct, namely, that the disease depends more upon unfavourable anatomical factors than upon unfavourable bacteriological factors, then the removal of the anatomical handicap and the provision of the wide open door into the general chamber of the nose must be regarded as the prime, and even perhaps the only necessity in the treatment.

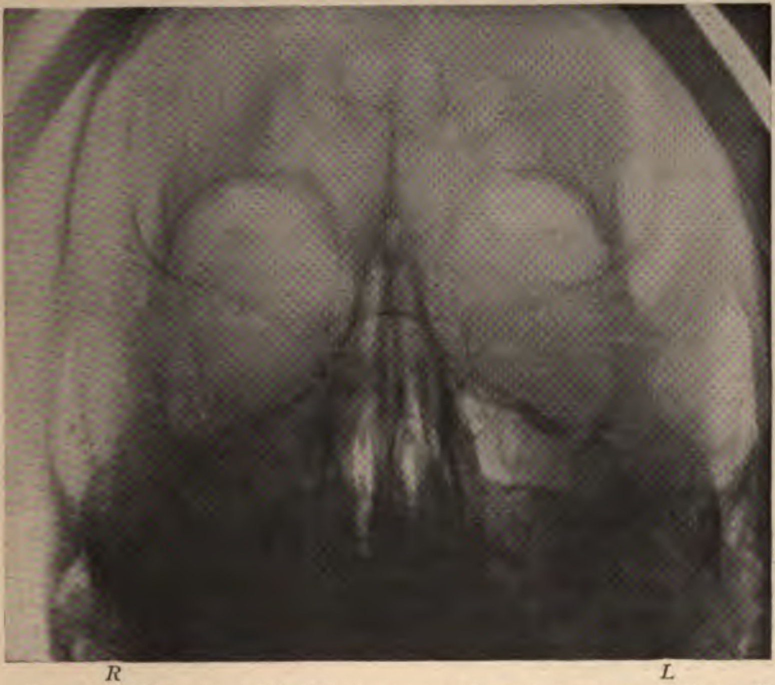


FIG. 101.—Skiagram of the face in suspected nasal sinus suppuration. The left antrum and frontal sinuses are clear and healthy. The right antrum is dull. (Skiagram by Dr. Ironside Bruce.)

But this removal of obstruction to drainage must, in certain cases and particularly in frontal sinus suppuration, necessitate, none the less, an external operation in order to clear away, not the entire lining membrane of the cavity, but such polypoid masses as are liable to block the drainage through the sinus orifice or duct even although that duct may have been enlarged.

In contrasting the value of the external with the intranasal operation, therefore, no general law applicable to all cases can be laid down. It was hoped, for example, that, as the trauma of an intranasal operation is less than that of an external operation, the special risks of the latter, that of extension of sepsis to the soft parts and to the bone of the sinus wall and cranium, would be avoided. But the hope has not altogether been realized, since diffuse osteomyelitis (see p. 372) has been known to follow the intranasal operation. Nevertheless, the main contention that the intranasal operation, *in skilled hands*, is the safer, we may, perhaps, admit.

The skilled hand, however, is an essential proviso. All of the intranasal operations on the sinuses demand an intimate knowledge of the anatomy and topography of the nose, as well as a delicate and instructed sense of touch on the part of the operator. Otherwise, disaster is certain to follow.

This leads us to the final principle of treatment, which is that **no operation should exceed in risk the disease it is designed to cure.**

In concluding these preliminary remarks, and in order to avoid giving a false impression of the value of nasal sinus surgery, we must emphasize the fact that in the vast majority of cases, complete and permanent recovery from the disease does take place, if only the case is efficiently and perseveringly treated. But perseverance, on the part of the surgeon, is necessary, and patience also on the part of his client, since the progress towards recovery is nearly always exceedingly slow, even after the whole of the diseased sinuses have been opened up and are draining, a consummation which usually entails a succession of three, four, five, or even more operations at intervals of weeks or months, according as first one, then another, then a third diseased cavity is revealed to the surgeon by the removal of the disease from their neighbours

We proceed now to deal with the sinuses in detail.

Clinically, the accessory sinuses of the nose arrange themselves into two groups :

1. The *anterior* group, which opens into the middle meatus below the middle turbinated body, and includes the frontal sinus, the anterior ethmoidal cells, and the maxillary antrum ; and
2. The *posterior* group, composed of the posterior ethmoidal cells and the sphenoidal sinus, which open above the middle turbinal into the superior meatus.

In typical cases of suppuration affecting either of those groups, pus from the anterior group appears in the middle meatus, oozing out from under the anterior end of the middle turbinal, and appearing at the anterior nasal orifice, while pus from the posterior group is directed by the middle turbinal, which forms the floor of the superior meatus, backwards into the naso-pharynx, where it may be seen, on posterior rhinoscopy, coming from above the posterior end of the middle turbinal.

The individual members of each of these groups are frequently associated with one another in suppuration, infection readily passing from one to the other.

THE ANTERIOR GROUP OF SINUSES

Chronic Suppuration.—*Symptoms.*—Amid the great variety of symptoms ultimately referable to suppuration of the anterior group of accessory sinuses, that of *muco-purulent discharge* from the nose is the most constant. This discharge is nearly always intermittent, and generally, but not invariably, passes out through the anterior naris. It varies in quantity and consistency. From the presence of bacteria of putrefaction, the pus is often fœtid, and the fœtor is experienced by the patient as well as by his friends. The *subjective fœtor* is a sign of some importance, for, as we have seen, in atrophic rhinitis, the fœtor is only objective.

As a rule, the discharge is most copious and the fœtor most marked in the morning. This arises from the circumstance that during the night, while the patient is in the recumbent position, the pus from the cavities flows slowly out of the ostia, and collects about the nasal passages, to be discharged in the morning when the head is upright.

It occasionally happens, however, that the patient fails to observe any nasal discharge or cacosmia, and comes complaining simply of nasal obstruction, which, on examination, we find to be due to the presence of hypertrophic rhinitis or of polypi, secondary to the prolonged irritation of the nasal mucous membrane from the continual bathing with pus. Or, it may be, sore throat and hoarseness are the phenomena which first attract the patient's attention. Again, we come across cases where the nasal disorders are quite unobtrusive, and the only complaint is headache, usually but not invariably frontal in situation. Apart from headache, pain may be present in the cheek, teeth, root of the nose, or supra-orbital ridge, according to the sinus affected, although here also no general rule can be laid down which is applicable to all cases. To begin with, suppurative sinusitis is quite frequently a painless disease; and secondly, in many cases, where pain does exist, its situation affords no clue whatever as to which particular sinus is affected.

Apart from these symptoms, the patient's chief or sole complaint may be one of a series of *secondary or reflex phenomena*, such as defective vision, lachrymation, orbital pains, giddiness, mental dullness and confusion, insomnia, tinnitus and deafness (from chronic Eustachian catarrh), etc.; while most patients suffer in their general health from the toxic absorption of septic products, this last depending usually upon whether or not the pus is obtaining free exit from the affected sinus or sinuses.

In most cases of sinus suppuration affecting the anterior group, more or less hypertrophy of the inferior turbinal is present, and quite commonly mucous polypi present themselves, sometimes in profusion, growing from the region of the middle turbinal. The presence of pus combined with multiple polypi may be regarded as an indication of purulent ethmoiditis (ethmoidal cell suppuration).

Where the ethmoid is free of disease, and there is little or no polypoid disease to disturb the relationship of the structures, pus may be seen emerging from underneath the middle turbinal, which, in these circumstances, frequently presents an appearance as if it were cleft or split from before backwards, the cleft being marked by a thin, yellow streak of pus. The turbinal is, of course, not actually split. What has happened to produce this appearance is, that the anterior lip of the hiatus semilunaris, into which several sinus-ducts open, having become swollen and inflamed from the purulent infection, has pushed the middle turbinal inwards towards the septum, and the apparent split is the interval between the lip of the hiatus and the middle turbinal.

This appearance invariably denotes suppuration in one or more of the anterior group of sinuses.

The next step in our investigation is to determine which sinus or sinuses of this group are affected; whether it is the maxillary antrum; the frontal sinus; or the anterior ethmoidal cells, or some combination of them. We proceed now to discuss these sinuses seriatim.

THE MAXILLARY ANTRUM

Anatomy.—The maxillary antrum is the largest of the nasal accessory sinuses. A thin-walled cavity of irregularly pyramidal shape, it extends from the lateral wall of the nose out to the malar bone; from the roots of the second bicuspid, and the first and second molar teeth up to the orbit, of which its roof forms the floor; and from the canine fossa in front to the pterygo-maxillary fossa behind.

The antrum opens into the nose by a small aperture situated in a circular or oval membranous diaphragm lying lateral to the anterior end of the middle turbinal, being the lowest of the orifices of the sinuses which open into the hiatus semilunaris, those of the anterior ethmoidal and of the frontal sinus lying above it. (Figs. 102 and 111.)

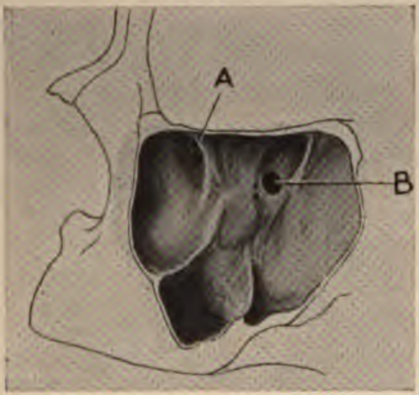


FIG. 102.—Maxillary antrum with the outer wall removed to show the level of the sinus ostia (after Hajek). *A*. The normal ostium which opens into the hiatus semilunaris. *B*. The accessory ostium (inconstant) which opens into the middle meatus of the nose above the inferior turbinal.

In a large percentage of cases, the antrum possesses also a second, or accessory orifice, a little below and behind the usual opening. But neither of these openings is situated low enough for draining the antrum in the erect posture. (Fig. 102.)

The close relationship of the antrum to the roots of the teeth exposes it to infection in dental caries, and, it is said, also in pyorrhœa, while its cavity is liable to be encroached upon by expanding dental cysts. Similarly, the ethmoidal series of cells, occupying a position contiguous to the upper and inner angle of the antrum, exposes the latter to the infection when the ethmoid cells are suppurating.

MAXILLARY ANTRUM SINUSITIS. (SUPPURATION OR EMPYEMA OF THE MAXILLARY ANTRUM)

Acute Antrum Sinusitis is a not uncommon complication of acute nasal catarrh, with or without simultaneous suppuration in the ethmoidal or frontal sinuses.

In the acute disease, the changes in the lining membrane of the cavity are relatively slight, and the simple washing out of the cavity is often sufficient to lead to cure.

Symptoms.—The complication is generally denoted by the persistence of nasal muco-purulent discharge from one or other or both sides after the other symptoms of the catarrhal attack have subsided. Stuffiness in the affected side of the nose is usual from sympathetic engorgement of the turbinals, and complaints of dullness and heaviness in the head, listlessness, lack of energy, and other signs of toxic absorption are common. Headache is usual, referred to the forehead, and neuralgia of the infra-orbital type or face-ache may add to the patient's sufferings. A pyrexial temperature is common, but many cases are entirely free from fever.

These last symptoms are most pronounced when owing to closure of the natural orifice the pus is retained in the antrum under pressure. (*Closed sinusitis.*)

On examination, signs of rhinitis are seen together with pus in the middle meatus as described at p. 324. But in closed antrum sinusitis, no pus or other evidence of sinus suppuration may be visible on rhinoscopy. (See also below for signs of pus in the antrum.)

Chronic Maxillary Antrum Sinusitis may be due to an acute attack which has been neglected ; or to gradual infection from the ethmoidal or other sinuses, in which case it is often associated with polypus formation in the nose. A fairly common cause, though not so common as is popularly believed, being responsible for only about one-third of the cases (Logan Turner and C. J. Lewis), is infection of the cavity from a septic bicuspid or molar tooth, and it has also been suggested that pyorrhœa alveolaris may lead to antrum sinusitis.

The pathological changes are those found in chronic sinusitis. (See p. 317.) Polypus formation within the cavity of an infected antrum is the rule when the disease is chronic, and the polypi may grow until the whole cavity is occupied by them.

The *Symptoms* of chronic maxillary antrum suppuration are those of chronic sinus suppuration in general. (See p. 323.) In this case the headache is usually frontal, and local pain is unusual though not unknown.

In all cases suspected of antrum disease, recourse is had to **transillumination** of the face.

Method.—A small electric lamp (5 to 8 volts), suitably protected, is inserted into the patient's mouth as he is sitting in a darkened room. Make sure that the lamp is well in the centre of the mouth, with the lips firmly closed round it. If the patient wears a dental plate, it should be removed before transillumination.

When the nose and its sinuses are healthy, the glow of the light proceeding from the lamp in the closed buccal cavity permeates more or less strongly every part of the face, nose and cheeks, and a red fundus reflex can be perceived in the eyes, both by the surgeon and by patient himself. The intensity of the glow varies in different individuals; in thin-boned, fair persons, the illumination is bright; in stout, large-boned, dark people, especially if the palate is narrow or highly-arched, or the nasal passages obstructed, the illumination is feeble or dull. A light of low brilliancy is therefore used for thin people, and a light of high brilliancy for stout, dark people.

As sinus disease is frequently unilateral, we can compare one side of the face with the other.

The particular areas of luminosity to be observed are those crescents of red light lying along the inferior margin of the bony orbit, and known as the "infra-orbital crescents." When this crescent is present, the antrum transillumination is called "clear"; when it is absent, antrum transillumination is "dull." The amount of light traversing the anterior wall of the antrum and showing on the cheek is very variable, but almost always it is much darker there than at the lower edge of the orbit.

Conclusions.—When the antrum transillumination is equally clear on both sides, the antra are empty and probably healthy; when dull, the antrum is filled more or less—it may be with pus, polypi, or a solid growth.

The following table summarizes the conditions which influence transillumination (Brown Kelly):

A. *Brilliancy is favoured* by—
   1. Cysts with clear contents, distending and thinning the antral walls.

B. *Brilliancy is decreased* by—
   1. Collections of mucus, blood, or pus in the antrum;
   2. Inflammatory and polypoïd thickenings of the antral walls; and
   3. Tumours in the antrum.

Fallacies.—If the pus in the antrum is small in quantity, the suppurating antrum may appear as clear as the healthy antrum.

On the other hand, there may be dullness on transillumination and yet no pus present in the antrum, in the case of

1. Unusual thickness of the bony walls, with a small, deeply seated cavity. This is a bilateral character as a rule,

consequently *bilateral dullness on transillumination bears less significance than unilateral dullness.*

2. Thickening of the antral lining membrane, in consequence of previous disease.
3. Solid tumours of the antrum.

In addition to transillumination, recourse should also be had, if possible, to *examination by X-rays.* (See also p. 318.)

If, then, in a case with purulent discharge from the nose, the antrum on one or both sides is dull, this cavity may either contain pus or be occupied by a solid growth.

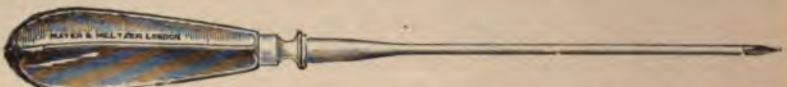


FIG. 103.—Lichtwitz Trocar and Cannula for proof-puncture of maxillary antrum.

The next step taken, therefore, is to ascertain the nature of the contents of the antrum. This step is—

Exploratory or Proof Puncture, which is usually performed with a very fine and long trocar and cannula. (Lichtwitz's, Fig. 103.) The puncture is made with cocaine-paste anæsthesia under the inferior turbinal and about half an inch from its anterior end. The point of the instrument, which is carried well above the floor of the nose, is directed upwards, outwards, and backwards, in a direction towards the upper margin of the auricle of the same side, and it is driven in with a jerk. In this act, the patient must keep his head erect and level. If he inclines it

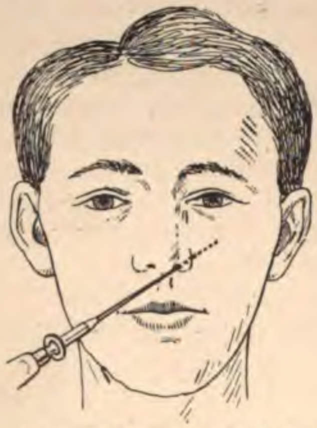


FIG. 104.—Proof-puncture (Lichtwitz) of the Maxillary Antrum. Insertion of trocar and cannula. Head erect.

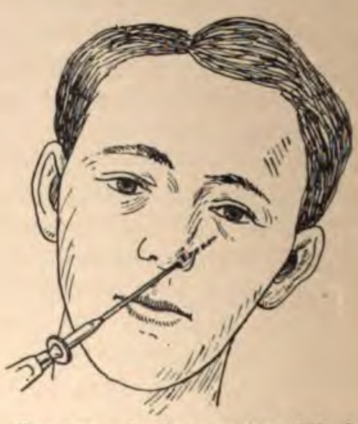


FIG. 105.—Faulty position of the head in making proof-puncture of the maxillary antrum, showing the danger of entering the orbit.

towards the same side, as he is apt to do, the needle may penetrate the orbit. (Figs. 104 and 105.)

In most cases there is no difficulty in entering the cavity, but it occasionally happens that when the antrum is small and deep-seated, the point of the Lichtwitz trocar misses the antrum, and passes instead into the soft tissues of the cheek. It is always wise, therefore, after puncture, to make sure that the point is in the antrum by gently moving the instrument about in a lateral direction.

The trocar is now removed, and a stream of warm, sterilized saline or boric acid solution is injected through the cavity by means of a suitable syringe, the patient holding his head forward over a basin to catch the fluid, which is then carefully examined for pus, etc. Finally, the cavity is perflated to expel all the fluid and the cannula is withdrawn.

Formerly, it was the practice to blow through the cannula (perflation) prior to syringing, but that is now abandoned, as it is possible in doing so to blow air into a vein and to cause dangerous symptoms.

The *Watson-Williams Syringe* provides us with an alternative method of testing the antral cavity. This is an aspirating syringe, with a long, sharp needle bent at a right angle. The needle is first inserted into the antrum, and the syringe, filled with normal saline, is fitted on to it. A little of the saline is syringed into the antrum, and then the piston is drawn back again so as to aspirate the liquid back into the syringe. If the antrum contains pus, the liquid returns turbid. If it is empty, air only is aspirated, or the fluid returns clear.

If when the attempt is made to draw back the piston, it is found difficult or impossible to do so, the reason is that the needle point is blocked, and in these circumstances, the likelihood is that it is a polypus inside the antrum that has blocked the needle-point.

In addition to effecting this relatively coarse test for pus, Watson-Williams also has the return fluid examined for bacteria even when pus is not found, and unless it is sterile, he does not consider the sinus to be healthy.

Assuming that the antrum contains pus, the next question to be answered is : Does the presence of this pus in the antrum mean actual disease there, or is the cavity only acting as a passive reservoir for the reception of pus draining into it from the frontal sinus or the anterior ethmoidal cells ? Perhaps the differentiation between the former condition, sinusitis, and the latter, empyema, is not a matter of much practical importance, since if the antrum has acted as a reservoir to the other cells for any length of time, infection of its walls is almost inevitable. The distinction may, however, be made, if necessary, by attention to the following points:

1. In simple empyema, after washing out the cavity transillumination will show the cavity to be clear, whereas in antrum suppuration, with the lining membrane thickened or polypoid, the cavity after washing out will remain dull on transillumination.

2. Bleeding from the cannula is highly suggestive of polypoid degeneration of the lining membrane.

These signs are, of course, only found in chronic disease.

Prognosis.—The prognosis of acute sinusitis of the antrum is good. Many cases get well spontaneously after simple lavage of the cavity, or failing that, simple nasal drainage is sufficient. The shorter the course of the disease has been, the better, naturally, is the prognosis.

The prognosis of chronic antrum suppuration, as such, is also good, relatively. That is to say, if it is allowed to remain untreated, it will persist indefinitely, and lead to serious sequelæ. But if the cavity is opened and drained, the disease will get well, gradually and slowly, and by fits and starts perhaps, but ultimately the patient will be cured.

Treatment.

Acute Suppuration.

1. The Lichtwitz puncture and lavage with sterilized normal saline repeated daily for three or four days is sufficient to cure early acute suppuration, if there are no deep changes in the mucosa. Indeed, it occasionally happens that a single lavage is all that is required.

Drainage of the cavity may also be furthered by amputating the anterior end of the middle turbinal. (See p. 339.)

2. If that fails, the case should be treated as one of chronic suppuration of the milder type, and nasal antrostomy performed. (See p. 331.)

While these proceedings are being carried out, the general purulent rhinitis should also be treated. (See p. 262.)

Chronic Maxillary Antrum Suppuration is treated by operation on the antrum, and there are two methods in vogue: (1) nasal antrostomy, or the making of an artificial opening or window between the nasal cavity and the antrum; and (2) the more radical procedure known as the Caldwell-Luc operation. The former usually suffices for the ordinary case, and the latter may be reserved for cases with much polypoid change in the antrum. It may also be employed if the nasal antrostomy fails.

Some operators, indeed, prefer the Caldwell-Luc for all cases, but it is a more severe operation, and in the writer's experience, has not proved to be any more efficacious than the simpler nasal antrostomy.

NASAL ANTROSTOMY

Description.—The intranasal formation of an artificial opening between the antrum and the nose.

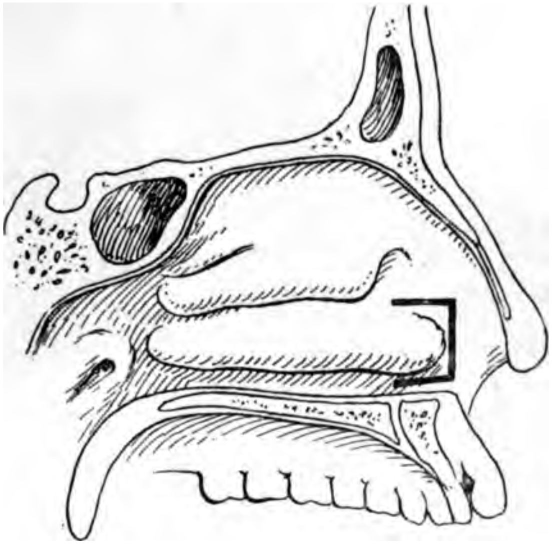


FIG. 106.—Nasal Antrostomy. Flap incision.

Indications.—The operation is often nowadays employed for all varieties of antrum suppuration, but it is of special value in mild and early cases.

Anæsthesia.—*Local,* by the application of cocaine-adrenalin paste, and 1 per cent. novocain solution injected into the antrum.

General. Chloroform, after one application of cocaine-adrenalin paste to the site of operation.

Operation.—The patient is lying on his back with head and shoulders raised to diminish hæmorrhage ; later in the operation, the head is turned to one side. The preliminary application of cocaine-adrenalin paste to the lateral wall of the nose is recommended as a hæmostatic. It should be applied just before the general anæsthetic is begun.

Under good illumination, a vertical incision is made into the mucosa of the outer wall of the nose in front of the anterior end of the inferior turbinal from a spot on a level with the attachment of the turbinal down to the floor of the nose. From both ends of this incision, horizontal cuts are made down to the bone of the outer wall of the nose above and below the turbinal. (See Fig. 106.) The flap thus marked out, which includes the anterior end of the turbinal, is undermined with a periosteal elevator, and removed either by snaring or by nasal forceps. It is frequently also necessary to cut through the attachment of the anterior end of the inferior turbinal to the wall of the nose with nasal shears.

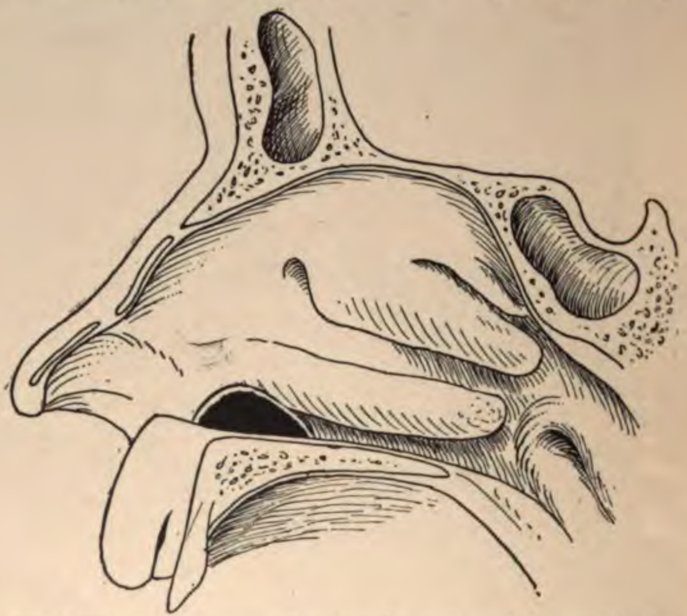


FIG. 107.—Nasal Antrostomy. Opening when the inferior turbinal can be spared (Claoué's operation).

Through the bone thus bared a burr is thrust into the antrum, the fragments being removed with nasal forceps, which can also at the same time be employed to enlarge the new opening to the desired size by bites from the posterior edge.

The anterior edge of the opening is smoothed down by burrs —Watson-Williams's frontal sinus rasps (Fig. 118) being very useful for the purpose.

The little finger is now passed into the nose and hooked through the opening into the antrum, in order to ascertain the size of the opening and the state of the antral cavity as regards polypi. Bent nasal forceps, or adenoid forceps (Fig. 134) are passed into the antrum to seize and remove polypi, and the antral walls may also be curetted lightly.

During these manipulations, bleeding is very free, but if a stout strip of sterile gauze is packed into the nose, so as to occupy the posterior naris, and the head is turned on one side, the blood will not run into the throat. If necessary, a sponge on a sponge-holder may be inserted into the naso-pharynx from the mouth, and held there. Packing the nose and antrum after operation is rarely required for hæmorrhage.

Modifications.—The classical nasal antrostomy consists in making the opening under the anterior end of the inferior turbinal, that bone not being interfered with unless it comes so low as to incommode the operator. (Fig. 107.)

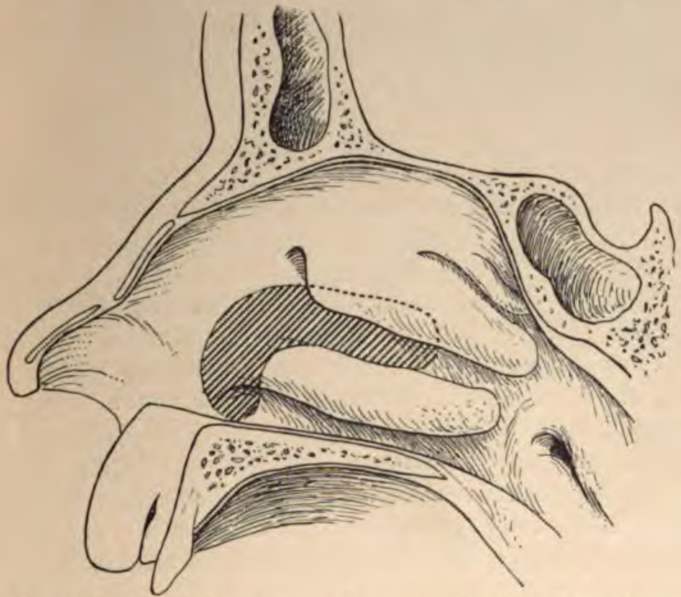


FIG. 108.—Nasal Antrostomy. Large opening into the antrum.

Experience, however, shows that during convalescence this small opening may altogether cicatrize over, and the foregoing is the operation performed and recommended by the writer in all save antrum sinusitis of quite recent origin.

In chronic suppuration with much polypoid disease of the antrum the operator need have no hesitation in enlarging the antrostomy opening backwards, keeping well above the inferior turbinal so as to preserve that body for physiological reasons. This carries the removal up towards the ethmoidal region. (See Fig. 108.)

Even this antrostomy opening will diminish in size during convalescence.

After-Treatment.—The antrum is washed out twice daily through an antrum tube passed through the opening into the cavity, sterile saline solution being used. The patient should be taught how to pass the tube and to use the syringe, which he goes on using until the discharge begins to lessen, when he should reduce the syringing, and finally stop it.

Results.—The above operation suffices for practically all cases of antrum suppuration. The writer finds the Caldwell-Luc operation very rarely necessary.

Muco-pus continues to be washed out of the antrum in gradually decreasing quantity for a year or eighteen months, being profuse only when the patient gets a " cold." Finally, it disappears.

THE CALDWELL-LUC OPERATION (THE "RADICAL" MAXILLARY ANTRUM OPERATION)

Description.—The opening into the antrum through the mucosa and bone of the canine fossa, in order to remove the diseased mucous lining, followed by the formation of a permanent opening in the nasal wall of the cavity, the route through the canine fossa being closed.

Indications.—Chronic antrum suppuration of long standing, especially if attended with polypus formation. It is also indicated if the nasal antrostomy operation fails.

Anæsthetic.—Chloroform with cocaine-adrenalin paste applied in the nose to the nasal wall of the cavity. The mouth is cleansed prior to operating by the removal of carious teeth, and by the use of antiseptic mouth washes. (See also p. 52.)

The patient lies on his back with head and shoulders raised.

The region of the mouth to be operated on is shut off by means of sterile gauze packed in between the teeth and the cheek, so as to keep the blood out of the pharynx. The mouth is slightly open, and the lip and angle of the mouth are held back with a suitable retractor.

The incision is made high up in the gingivo-labial recess, and opens up the canine fossa for about three-quarters of an inch. With an elevator, the periosteum is raised off the bone upwards, stopping short of the infraorbital nerve, injury to which will induce post-operative neuralgia.

With gouge and mallet, a disc of the thin, hard bone of the anine fossa is raised, giving entrance to the antrum, and the pening thus made is enlarged as may be necessary with cutting bone-forceps. (Fig. 109.)

The cavity of the antrum having been emptied of pus by means of long strips of gauze, the interior is inspected with head-light and mirror. Polypi are removed with nasal forceps, and, if necessary, the mucosa may be lightly curetted. This step is attended with profuse hæmorrhage, which will stop, however, as soon as the evacuation of polypi and turgid mucosa is completed.

The next step is the formation of a nasal opening, the situation of which should correspond to the wall of the bone beneath the inferior turbinal if there is sufficient room here for it. Otherwise, some of the turbinal must be sacrificed, though as sparingly as possible. The nasal opening should be large enough to admit the forefinger.

The wound in the buccal mucosa may now be closed with a couple of catgut sutures, and if a packing is required for hæmorrhage, it should be inserted into the cavity through the nose.

After-Treatment.—The operation is more severe than simple nasal antrostomy, there being more shock and pain, while the cheek may swell, and the eyelids become œdematous. For this, hot fomentations give relief, and in any case it subsides in a few days.

A slight, though so far unestimated risk of septic pneumonia attends operations on a suppurating antrum. It may be due to septic venous thrombo-phlebitis, or to the antral discharges finding a way into the lungs at the operation; this latter accident may be avoided by lavage of the cavity, through a Lichtwitz tube before the operation is started.

The *Results* as regards the antrum suppuration and polypus formation are good.

During the months of slow convalescence, the trying symptom sometimes appears of severe facial neuralgia. It is probably due to the action of scar-formation on nerve-endings. It may last several months, but eventually disappears, and its departure may be hastened by m.20 of Easton's Syrup internally three times a day.

The *alveolar operation*, through a tooth-socket on the maxillary antrum, is seldom performed by modern rhinologists, but it is of prime importance to have any carious or septic tooth removed which is suspected of infecting the antrum. The roots of the second bicuspid, and of the first and second molars, are those which come into closest relationship with the cavity.

Comparative Value of Operations.—In comparing nasal antrostomy as described above with the Caldwell-Luc operation, the greater removal of the inferior turbinal advised in the former must not be overlooked, as the more this structure is removed, the greater is the likelihood of subsequent rhinitis sicca and crust-formation. At the same time it is necessary to add that the success of both operations on the antrum disease seems to depend largely upon the size of the nasal opening, and a nice problem is presented in every operation as to how much should be removed. In other words, the comparative value of the operations is not yet finally settled.

THE ETHMOIDAL CELLS

consist of six or eight small cavities occupying the upper region of the nose, extending from the bulla ethmoidalis, and sometimes from the region of the fronto-nasal duct (infundibulum) in front to the anterior wall of the sphenoidal sinus behind (Figs. 111 and 112), and occupying the space between the middle turbinal medially and the inner wall of the orbit laterally.

Those of their number which are contiguous to the infundibulum, known as the *fronto-ethmoidal cells*, may extend up and enter into relationship with the frontal sinus. Behind the fronto-ethmoidal cells lie others of the *anterior ethmoidal group* opening also into the hiatus semilunaris, and lying in relationship with the infundibulum as well as with the lacrymal bone and the papyraceous bone of the inner wall of the orbit. We have already drawn attention to the occasional presence of an ethmoidal cell in the middle turbinal, and it is said that a cell is even sometimes present in the crista galli. Posteriorly come the *posterior ethmoidal cells*, usually only two in number, and larger individually than the members of the anterior groups. They open along with the sphenoidal sinus into the superior meatus above the middle turbinal, and lie in front of the sphenoidal sinus, medial to the inner wall of the orbit near to the optic foramen.

The surface and other limits of the ethmoidal labyrinth are as follows :—

Medially, the operator should never transgress the limits of the attachment of the middle turbinal to the roof of the nose; otherwise, he comes into the region of the cribriform plate. The level of the cribriform corresponds with the transverse limb of the T-shaped suture at the root of the nose which the ascending process of the superior maxilla and the nasal bone form with the nasal process of the frontal bone (see Fig. 109), and this marks the upward limit of operations on and medial to the middle turbinal.

The outer, or orbital landmark of the ethmoidal labyrinth, lies slightly medial to the inner margin of the orbit from the T-shaped suture to the lower end of the lacrymal bone. This limit deviates outwards as we pass to the posterior ethmoidal region in conformity with the trend of the medial wall of the orbit. (Fig. 109.)

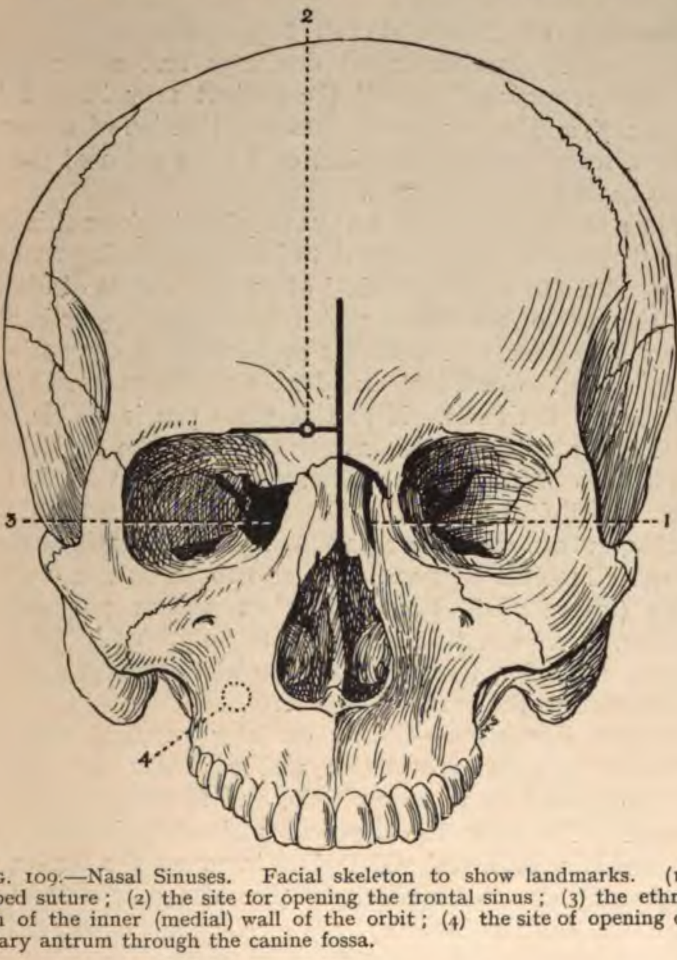


FIG. 109.—Nasal Sinuses. Facial skeleton to show landmarks. (1) The T-shaped suture; (2) the site for opening the frontal sinus; (3) the ethmoidal section of the inner (medial) wall of the orbit; (4) the site of opening of the maxillary antrum through the canine fossa.

The ethmoidal cells are separated from the orbit and from the lacrymal recess by very thin wafer-like bone which is easily perforated by disease and operative manipulation, but, so long as the stout orbital fascia remains intact, orbital cellulitis will not occur. Here also the cells enter into relationship with the antrum.

The importance of the ethmoidal cells in accessory sinus suppuration depends upon three factors: (1) their liability to infection; (2) their liability to polypus formation; and (3) their liability to infect the outlying sinuses, the antrum, frontal sinus, and sphenoidal sinus.

Symptoms of Ethmoidal Cell Suppuration.—In its earlier stages, beyond purulent nasal discharge and a tendency to frequently recurring "colds," the patient may have little to complain of, and in many such cases the attention of the surgeon is only directed to the ethmoidal cells after some of the other sinuses have become infected.

If seen in this early stage, no pus may be visible on rhinoscopy, unless the examination happens to be made during an exacerbation. But the middle turbinal often shows signs of disease in being the seat of an œdema which frequently approximates to polypus formation, and a similar appearance of congestion and œdema is often to be detected under the middle turbinal affecting the region of the bulla. If at this place pus is visible, and if frontal and maxillary sinus suppuration can be excluded, the diagnosis of ethmoidal suppuration may be made with certainty.

Occasionally it happens that the amount of pus secreted is considerable, and can always be seen clinging about the middle meatus.

Some ethmoidal suppuration is also to be seen in ozænatous rhinitis (see p. 282), but it is then seldom progressive in character, it does not induce genuine polypus formation, and it does not require, therefore, any special treatment apart from that directed to the primary disease.

Polypus Formation.—Sooner or later purulent ethmoiditis is attended by polypus formation, and it is characteristic of such polypi to grow fast, to be multiple, and to recur rapidly when removed. The growth of polypi ultimately breaks up the ethmoidal labyrinth, and still further increases the tendency for the other sinuses to be affected. (See also p. 270 for further remarks on Nasal Polypus.)

Treatment of Ethmoidal Suppuration.

1. The simpler cases of the early stages may be cured by nasal douching, as by

℞. P. Potass. Chlor.
P. Sod. Bibor.
P. Sod. Bicarb. *aa.* ʒiv.
P. Sacch. Alb. ℥i.—M.

Four drams of this powder, dissolved in one pint of warm water, are syringed into the nose twice daily. After the lavage, the nose is blown, and a spray of

℞. Menthol,	gr. ii.
Ol. Eucalypt.,	♏iii.
Paraff. Liq. ad.,	℥i.—M.

used with an atomizer.

2. Such simple treatment failing, and in cases of greater severity where the structures about the middle meatus show œdema, swelling, and perhaps purulent secretion, operation is needed.

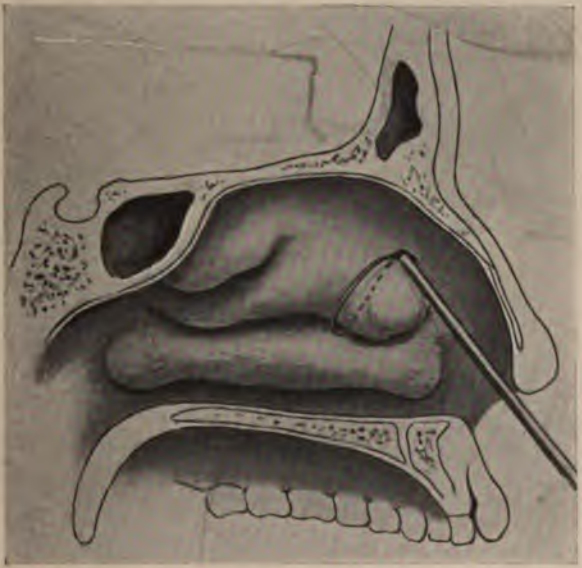


FIG. 110.—Removal by snare of the anterior end of the middle turbinal.

(a) In a minority of cases where suppuration can be definitely located to one or two cells, it may be possible, by means of Hajek's hook, used under cocaine-adrenalin anæsthesia, to open up and to drain the suppurating cells.

(b) Otherwise, when there is œdema of the middle turbinal, the anterior end of that bone should be removed, and perhaps the adjoining bulla, if swollen, opened by Hajek's hook or a curette. (See p. 278.)

Removal of the Middle Turbinal.—1. Under local anæsthesia, either by cocaine-adrenalin paste or by tampons, the bony attachment of the middle turbinal is cut through by means of nasal scissors from before backwards, the extent of this incision being dependent upon the amount of the bone to be removed. A snare is then passed into the nose and over the detached anterior end of the turbinal, the point of the instrument being inserted into the cut just made, while the wire loop passes around under the inferior surface of the bone. The snare is then quickly closed, and cuts through the bone. (See Fig. 110.)

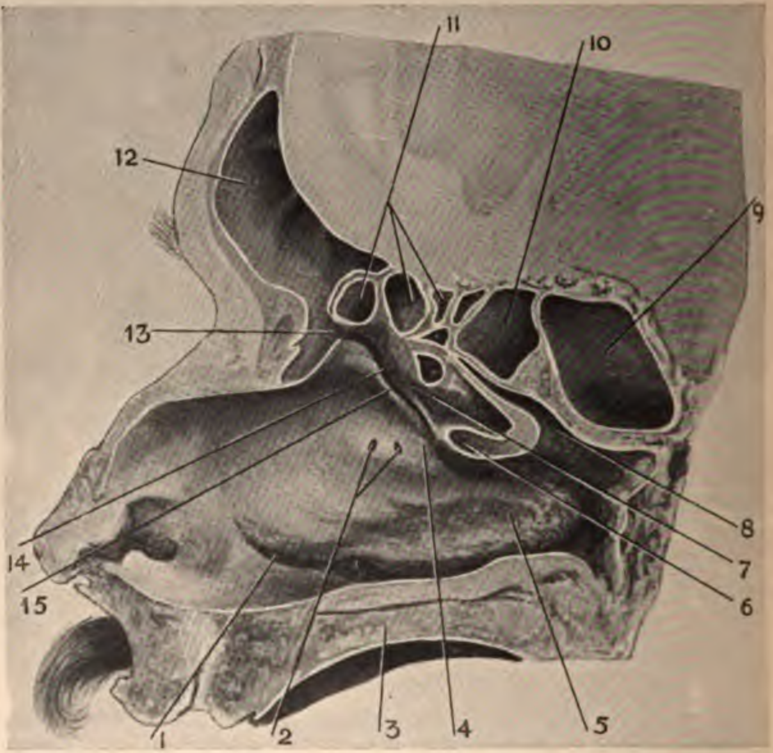


FIG. 111.—Sagittal section of the nose carried through the ethmoidal region. (After Onodi.) See also Fig. 112. (1) The inferior meatus. (2) Accessory orifices of the maxillary antrum. (3) The palate. (4) The middle meatus. (5) The inferior turbinal. (6) Stump of the middle turbinal, the remainder having been removed. (7) The bulla ethmoidalis. (8) The superior meatus. (9) The sphenoidal sinus. (10) A posterior ethmoidal cell. (11) Anterior ethmoidal cells. (12) Frontal sinus. (13) The fronto-nasal duct, or infundibulum. (14) The hiatus semilunaris. (15) The uncinate process. (The section is more lateral than that of Fig. 112.)

2. Or the anterior end is seized by the author's nasal forceps (Fig. 94) and twisted off. As much or as little of the bone as is desired may be removed by this simple method.

3. In cases with extensive polypoid disease, after the growths have been cleared so as to expose the field, the operation of curetting is necessary, as described in the section on nasal polypus. (See p. 278.)

The course of chronic ethmoidal suppuration is, like that of all nasal sinus suppuration, tedious, and it is usually necessary to warn the patient that several surgical interventions may be required before treatment can be left in his own hands.

It is necessary also for the surgeon to assure himself that the other sinuses are not involved, or do not become involved in the course of the ethmoidal disease, and if they do, to deal with them accordingly.

After the ethmoidal cells, assuming that they alone are affected, have been curetted or otherwise opened up, a longer or shorter period of slow suppuration ensues, but ultimately the swollen mucosa shrinks, the discharge dries up, and the orifices of the cells become visible as small openings in the vaulted sides and roof of the nose, the turbinal and other anatomical structures having disappeared as a consequence of the disease or the treatment. At this late period, in extensive cases, the patient is liable to become afflicted with rhinitis sicca. (See p. 280.)

For this reason, it is always advisable so to operate on these cases as to remove the minimum amount of turbinal and ethmoidal bone. It is wiser to proceed by small steps than by great and spectacular advances at the expense of tissue that may, with a little patience, recover itself, and resume its normal functions. Thus, while we do not hesitate to curette the ethmoidal region when it is the seat of multiple polypi, we postpone turbinectomy, and such destructive measures, until we are sure that the more conservative procedures of lavage, or simple cell opening have failed.

THE FRONTAL SINUS

Anatomy.—The frontal sinus is extremely variable, both in size and disposition. (See Figs. 100, 101, 111, 112.) It may be entirely absent, the infundibulum ending blind in the frontal bone. Or, at the other extreme, the sinus cavity may extend as high up as the frontal eminence, as far out as the external angular process of the frontal bone, and as far back in the roof of the orbit as the optic foramen. Extensions of the ethmoidal cell system, again, may project up alongside the infundibulum and come into relationship with the floor of the frontal sinus.

Moreover, it is usual for the frontal sinuses to be asymmetrically disposed. The sinus of one side may extend across the middle line of the forehead, and occupy a position which brings it partly in front of or behind its neighbour. In such conditions, the intersinus septum is extremely deviated, and the sinus of one side may be considerably larger than the sinus of the other side. Further, a perforation of this septum is by no means rare, so that disease in the one sinus will quickly pass to infect the other, and when one cavity is washed out through the cannula, the liquid returns by both nostrils.

The variability of the frontal sinus renders it necessary always to obtain an X-ray photograph before operating upon it. (Figs. 100 and 101.)

The *infundibulum* or fronto-nasal duct descends from the inner angle of the floor of the sinus in a downward and slightly backward direction, traversing the fronto-ethmoidal cell region to reach the hiatus semilunaris where it opens into the cavity of the nose. (Figs. 111, 112.) Sometimes, however, the duct ends in an ethmoidal cell, in which case frontal sinus secretion or discharge can only reach the nose after it passes through the cell. An ethmoidal cell (or cells) of much importance in the intranasal frontal sinus operation is that known as the *agger cell or group of cells* which occupies a position in the lateral wall of the nose anterior to the hiatus semilunaris, and in front of the infundibulum. (Fig. 112.) The importance of this group lies in the fact that when broken down by the curette, it gives ready access to the infundibulum at a much higher level (and so nearer to the frontal sinus itself), than can be obtained from the hiatus semilunaris.

External Guide to the Infundibulum. The duct lies exactly behind the junction of the transverse with the perpendicular line of the T-shaped suture at the root of the nose. (P. 336 and Fig. 109.) Behind the duct is the cribriform plate, and the anterior wall of an anterior ethmoidal cell.

Towards the roof of the nose the duct is separated from the olfactory fissure by a thick plate of bone.

The frontal sinus is in relation (a) through its floor with the orbit, of which indeed it forms part of the roof; and (b) through its posterior wall with the frontal lobe of the brain and its covering of dura. (Figs. 112 and 114.)

ACUTE FRONTAL SINUSITIS

The remote and isolated situation of the frontal sinus, and the length and fineness of its duct render its symptoms in acute inflammation more severe than is the case with any of the other

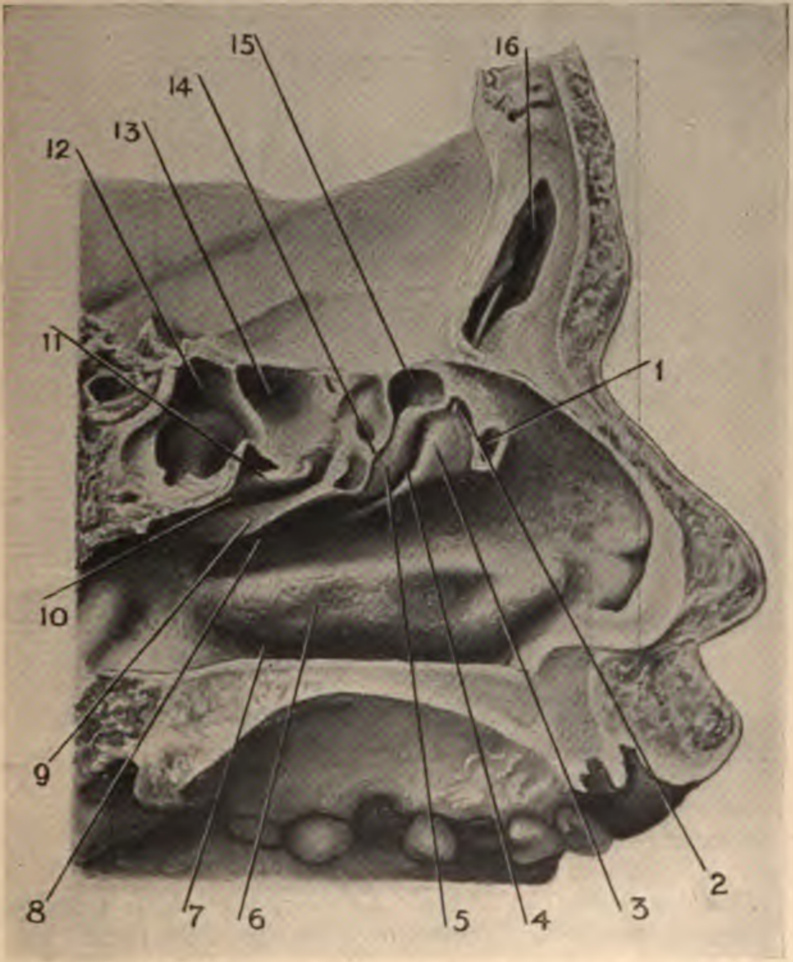


FIG. 112.—Sagittal section of the nose to the left of the middle line. See also Fig. 111. (1) Ethmoidal (agger) cell. (2) Infundibulum. (These form the key to the intranasal frontal sinus operation.) (3) Uncinate process. (4) Hiatus semilunaris. (5) Bulla ethmoidalis. (6) Inferior turbinal body. (7) Inferior meatus. (8) Middle meatus. (9) Posterior end of truncated middle turbinal. (10) Superior meatus. (11) Superior turbinal. (12) Sphenoidal sinus. (13) Posterior ethmoidal cell. (14) Recess of the bulla. (15) Anterior ethmoidal cell. (16) Frontal sinus. (After Onodi). (The section is more medial than that of Fig. 111.)

accessory sinuses. Moreover, acute frontal sinusitis is more dangerous to life by reason of the close proximity of the sinus to the brain, while the disease may end in considerable facial deformity if there is much bone destruction. (Fig. 113.)

Two varieties of the disease are distinguished: *simple acute sinusitis*, when the disease is confined to the sinus cavity; and the so-called *sinusitis exulcerans*, when one or more of the bony walls are attacked and undergo softening and cario-necrosis with extension of the suppuration of the soft parts beyond the limits of the sinus.

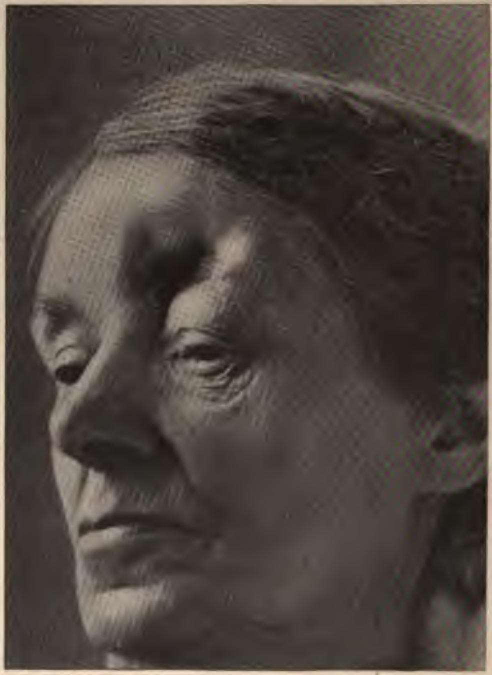


FIG. 113.—Disfigurement due to acute frontal sinusitis (exulcerans) with extensive bone destruction.

Symptoms.—In *simple acute sinusitis*, the chief symptom is pain, deep-seated and severe, over the site of the frontal sinus with marked tenderness on pressure, particularly on the bone of the roof of the orbit towards its inner angle, directly below the inner end of the eyebrow. This sign is most strikingly elicited by direct percussion with the finger tip on this spot.

Constitutional phenomena such as pyrexia and malaise, are usual. The patient is irritable and restless, and sometimes experiences mental disturbances, such as loss of memory and confusion of thought, without there being necessarily any brain involvement.

It is remarkable that nasal discharge may be entirely absent in acute frontal sinusitis, and that the interior of the nose may be quite devoid of trace of pus or other sign of disease. In most cases, however, swelling of the middle turbinal, or of the bulla ethmoidalis, with or without polypi, is present.

Simple acute sinusitis may spontaneously recover with a discharge of the retained pus through the infundibulum into the nose ; or it may be relieved or cured artificially ; or it may go on to the second type of the disease with or without an intracranial extension ; or, finally it may become chronic.

Sinusitis Exulcerans.—In this condition the sinus infection traverses one or other of the bony walls of the sinus, and infects the soft parts. The floor of the sinus is the most frequently attacked, and an abscess forms which discharges or is opened through the upper eyelid. Orbital cellulitis is prevented by the interposition of the orbital fascia. When the posterior sinus wall is destroyed by the disease, extra-dural abscess, meningitis, or abscess of the frontal lobe may occur ; and any of these different extensions may appear in combination.

The *symptoms* are those of simple acute sinusitis with the addition of the signs of abscess in the soft parts, but the extension may take place very early in the course of the disease.

When the anterior wall or the floor is attacked, the signs of abscess are unmistakable, there being great local swelling, œdema, pain, and tenderness, with pyrexia, until the abscess bursts or is opened. But when the redness and swelling appear in the eyelid, the true nature of the cause is seldom suspected. It is generally diagnosed as orbital cellulitis. When the posterior wall is attacked, however, no symptoms may be produced of a nature to indicate the occurrence of this accident, unless meningitis is set up.

The signs of extra-dural abscess in this situation have nothing to distinguish them from simple acute frontal sinusitis itself, and **abscess in the frontal lobe of the brain** is also usually latent, this being a " silent " region of the cerebrum.

Such symptoms as may exist consist of vague cerebral phenomena such as loss of memory, slow cerebration, and, more rarely, mental disturbances of a maniacal character. Occasionally, encephalitis surrounding the pus collection in the brain may reach as far back as the motor areas, and induce paretic or paralytic phenomena. Epileptiform convulsions sometimes attend the inception of the abscess. Or, as in otogenic brain abscess, the first and only indication of any cerebral complication may be the onset of coma. A usual event is for the abscess to be discovered accidentally at operation or *post mortem.*

Diagnosis.—Acute frontal pain and even tenderness may be caused by antrum suppuration, the frontal sinus being healthy. It is well, therefore, to exclude the antrum by puncturing and washing it out. Both sinus and antrum, however, may be affected.

It is a clinical rule, also, in all cases suspected of frontal sinus suppuration, whether acute or chronic, to have a skiagram of the skull taken by an expert radiologist, as pain over one eyebrow may be due to frontal sinusitis of the opposite side. (Figs. 100, 101 and 115.)

Prognosis.—If promptly operated on, the prognosis both of simple sinusitis and of the necrotic variety is good, so long as there is no intracranial extension. The cases recover. But the prognosis of frontal lobe abscess is grave, as meningitis is prone to follow its evacuation.

Treatment.—(a) *Simple acute frontal sinusitis* may be relieved or cured, for the time being, at any rate, by simple measures, all of which should be tried before proceeding to the major operations.

1. It is sometimes possible, after anæsthetizing the region of the hiatus semilunaris, to pass the frontal sinus cannula into the cavity, and to give immediate relief by washing it out with warm saline or boric acid solution. (See p. 351 for method.)

2. The anterior end of the middle turbinal may be amputated to relieve pressure and œdema about the orifice of the infundibulum in the hiatus semilunaris. (See p. 339.)

If these fail, the intranasal frontal sinus operation should be performed under a general anæsthetic (see p. 353), this proceeding being sufficient to cure all uncomplicated cases.

If, however, the intranasal operation has been performed and the pain or the headache or the pyrexia still persists, or, apart from this operation, if cerebral symptoms exist, the likelihood of the presence of an intracranial complication should lead to immediate external operation.

(b) *Sinusitis Exulcerans.*—In the presence of abscess of the soft parts external to the frontal sinus, no time should be wasted on intranasal treatment, whether operative or non-operative.

The abscess should be opened under a general anæsthetic, the incision passing through the opening in the skin if the abscess has already broken. After the pus has been evacuated, the fistulous opening into the frontal sinus is sought for, the skin ncision being extended, if necessary, to give access, and after it has been found, the soft and carious bone in its vicinity is all entirely removed. This usually means making a free opening into the frontal sinus, the interior of which may be inspected, cleared of pus, débris, and granulations, but without any curetting.

No further attempt at radical cure of the disease is made on this occasion. The sinus and the raw bone edges are rubbed with B.I.P.P.; the interior of the sinus is filled loosely with gauze; the skin wound, if extensive, may be partially united with stitches, leaving ample room for drainage, but nothing further is done, the object of the operation being merely the opening and draining of the abscess and the sinus.

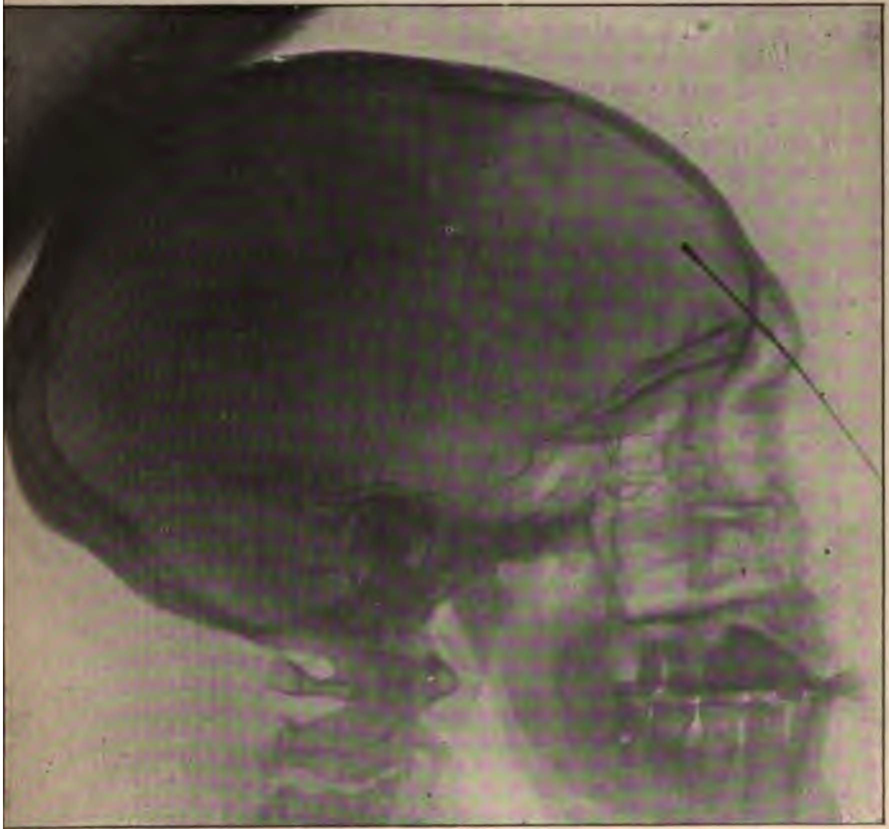


FIG. 114.—Frontal lobe abscess due to frontal sinusitis. The skiagram shows a probe in the abscess cavity. (The patient recovered and fought in the Great War.) (Skiagram by Dr. Ironside Bruce.)

The reason for this conservative attitude is that the presence of virulent sepsis favours the occurrence of spreading osteomyelitis (see p. 372), and no great bone operation should be undertaken until the acute inflammatory reaction has subsided. At a later date, however, when the wound has begun to granulate, it is re-opened, and the frontal sinus operation completed, as in the Ogston-Luc operation. (See p. 357.)

After-Treatment.—Boric acid fomentations are applied until the swelling subsides and the wound begins to granulate, and then the operation may be completed.

Watch should be kept for signs of extending osteomyelitis. (See below, p. 372.)

Abscess of the Frontal Lobe of the Brain.—If *discovered during the operation* on the frontal sinus as by a discharging sinus leading into the abscess cavity in the brain, the posterior wall of the sinus is removed to facilitate drainage, and a drainage tube inserted into the abscess-cavity. (See Fig. 114.)

In exploring for abscess, the dura should be exposed by removal of the posterior sinus wall, and if the symptoms are not urgent, the opening of the dura and the exploration of the brain should be postponed for two or three days, so as to permit meningeal adhesions to form. Exploration is conducted in the same way as for otogenic brain abscess, the depth of the search being limited to one inch lest the lateral ventricle be opened.

(If in the course of operation on frontal sinus, whether for acute or chronic disease, the dura is accidentally wounded, the wound if small should be freely exposed by removal of the bone, and, if necessary, enlarged, a strip of iodoform gauze being inserted for drainage between the lips of the dural wound.)

After a frontal brain abscess has been opened and drainage tube or tubes inserted, the outer wound is loosely packed with iodoform gauze, and the skin incision partly sutured, enough of a gap being left for free drainage and manipulation of the tube. The outer wound is dressed with warm boric acid fomentations.

The *after-treatment* follows the same lines as in otogenic brain abscess. (See p. 547.)

CHRONIC FRONTAL SINUSITIS

The long and tortuous infundibulum is responsible for the frontal sinus, once it becomes infected, remaining diseased for prolonged periods, and as the duct under those conditions is liable to periodic obstruction from œdematous swelling of its lining, the phenomena of pus-retention are very frequently manifest in frontal sinus suppuration.

The pathological changes within the sinus are similar to those in the antrum and elsewhere, namely, polypoid swelling of the mucosa, which may proceed until the cavity is filled with polypi. As with the acute disease, so with the chronic, the frontal sinus more frequently shows signs of implication of its bony walls than do the other sinuses, in the shape of erosion, and in rare cases of a spreading osteomyelitis of the cranium. (See p. 372.)

Symptoms.—In addition to the discharge of pus from the nose, and to the symptoms of polypi in the nose, which appear when the frontal sinusitis is combined with ethmoidal disease, the frontal sinus disease may set up several symptoms which are distinctive.

Local pain and tenderness, for example, coming and going according as the pus is retained or discharged is not uncommon, and with, or indeed without it, intermittent headache, paroxysmal and severe, is often experienced, either general in distribution or limited to the region supplied by the corresponding supraorbital nerve. Besides these phenomena, patients often complain of cerebral disturbances, such as mental lethargy, confusion of ideas, loss of memory; or of vertiginous attacks, and perhaps sickness and vomiting.

On the other hand, again, frontal sinus suppuration on one or both sides is often unsuspected, not giving rise to any symptoms whatever, and this is most frequently met with when sinusitis elsewhere has led to serious polypoid disease in the nose. So that it is only after the polypi have been removed, and the ethmoidal region has been cleared that the presence of frontal sinus suppuration is disclosed by the continuance of purulent discharge.

The appearances, on examination of the nose, vary according as the frontal sinus suppuration is or is not accompanied with suppuration in the ethmoidal and other sinuses. (In point of fact, frontal sinus suppuration is nearly always accompanied by signs of ethmoidal suppuration.)

When the frontal alone is affected, if pus is seen on examination it will be found emerging from under the middle turbinal, as in antrum suppuration, the frontal like the antrum being one of the anterior group of sinuses. We can observe this difference between them, however:—when pus is coming only from the maxillary antrum, the flow will be encouraged by the patient lowering the head; in frontal sinus suppuration, lowering the head makes no difference to the flow, but if the pus is wiped away from the middle meatus when the patient is sitting upright, it will appear again after a short interval. A purulent discharge appearing in this situation and behaving in this fashion, proceeds either from the frontal sinus or from the anterior ethmoidal cells, or from both.

Transillumination, when the frontal alone is diseased, will generally show the antrum to be clear. (Transillumination of the frontal sinus is unreliable.)

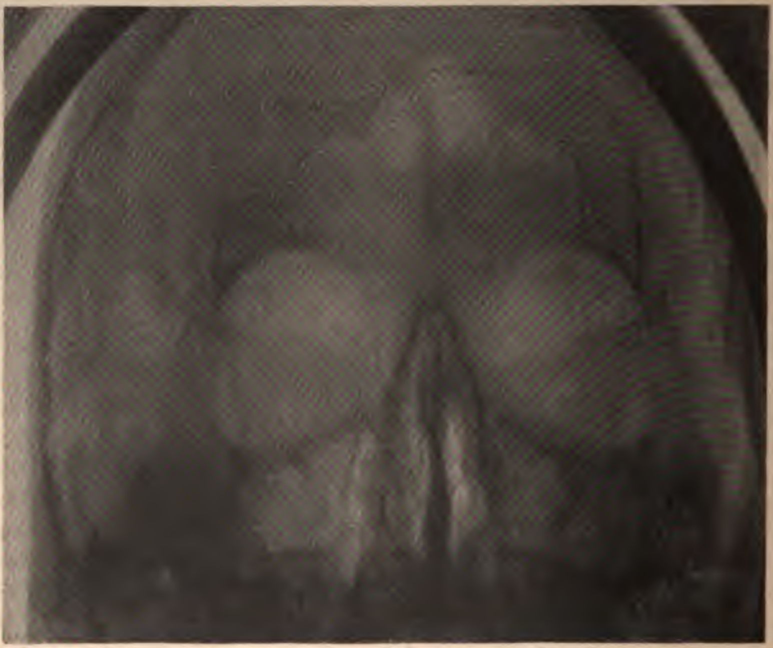


FIG. 115.—Clouding of the left frontal sinus with blurring of the outlines in sinus suppuration. The left maxillary antrum is also dull. (Skiagram by Dr. Ironside Bruce.)

Under those conditions recourse is had to X-ray examination and to lavage of the frontal sinus by a cannula inserted into the sinus through the nose and infundibulum (fronto-nasal duct).

By X-ray examination (see Figs. 100, 101, 115), the sinus, when healthy, is seen to be clear of shadow, while its outlines are distinctly marked and traceable in the skiagram. When, on the other hand, it contains pus, the sinus throws a shadow which renders the outlines of the cavity blurred and indistinct. (Fig. 115.) X-ray examination in suspected frontal sinus disease is indispensable not only as a means of diagnosis, but as the only means we possess of revealing the size, shape, and disposition of this extremely variable cavity, information which we must obtain before undertaking any operative treatment.

Treatment.—Lavage of the Frontal Sinus.—The catheterization of the fronto-nasal duct is sometimes quite easy and simple even with the middle turbinal and the ethmoidal cells undisturbed; at other times, and more commonly, the cannula cannot be inserted until operation has laid bare the hiatus semilunaris, and perhaps also, the fronto-nasal duct above its normal orifice.

When ethmoidal polypi are present, these must, of course, be first of all removed, and the site of their origin curetted (see p. 278), after which the insertion of the frontal sinus cannula presents, as a rule, no difficulties.

If polypi, or other signs of ethmoidal disease are absent, and the cannula cannot be passed, the anterior end of the middle turbinal may be removed, so as to expose the hiatus semilunaris; or what is better, the fronto-ethmoidal cells in front of the hiatus are opened with a curette, so as to afford access to the infundibulum at a spot superior and anterior to its normal orifice and more within reach of our manipulations. This last is, indeed, the first step in the intranasal operation on the frontal sinus. (See p. 353.)

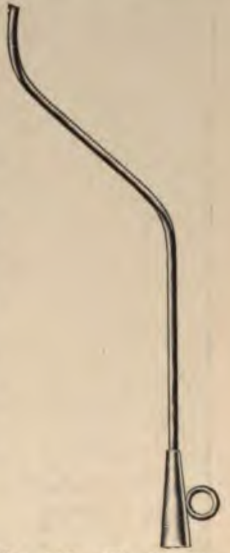


FIG. 116.—The Fronta Sinus Cannula.

Whatever plan be adopted, once the duct is made accessible, a frontal sinus cannula such as that here figured (Fig. 116), made of ductile metal so that it can be bent as desired, is passed into the nose under inspection, and so into the orifice of the fronto-nasal duct on the outer wall of the nose, but higher and further forward than one usually expects to find it.

Once the point of the instrument is engaged in the infundibulum, it passes freely and easily upward on lowering the outer end. By placing a probe or cannula outside the nose in a position corresponding to that occupied by the cannula inside the nose, the situation of the point of the latter can be approximately determined. When the cannula is *in situ*, the sinus is perflated, while the observer watches the escape of the secretion into the nose. If pus is blown out, we may wash out the cavity with a mild antiseptic lotion; but if no pus is obtained, it is safer not to douche out the sinus.

It must be said that although in most cases the point of the cannula, when the straight portion is in contact with the patient's upper lip, is in the frontal sinus (Fig. 117), nevertheless there are cases where this appearance is deceitful, and where it is lying in a high fronto-ethmoidal cell only.

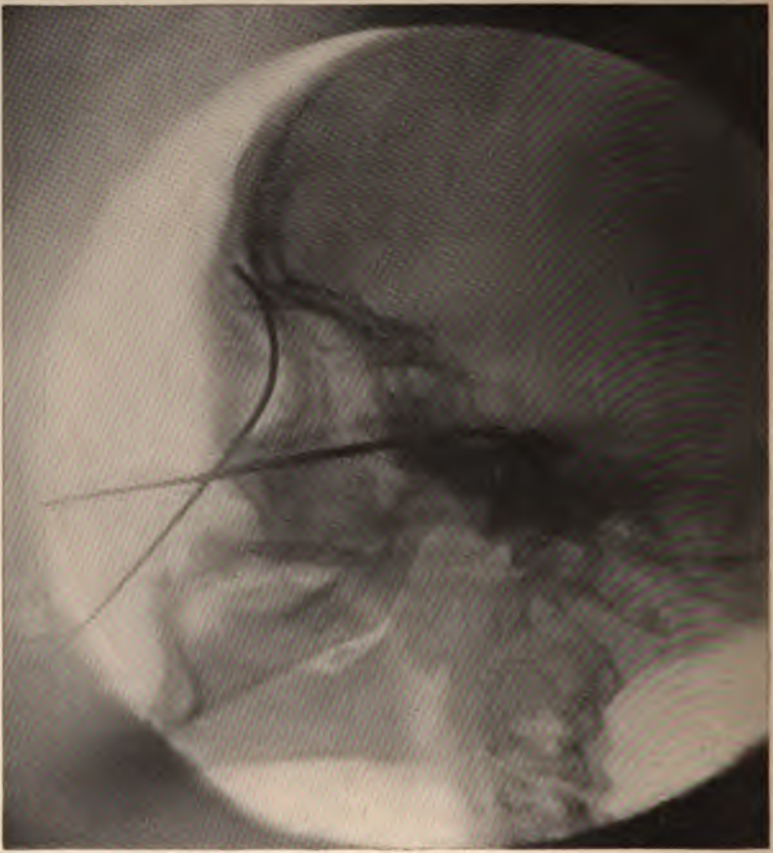


FIG. 117.—Skiagram by Dr. Robert Knox showing cannulæ in the frontal and in the sphenoidal sinuses.—Case of Mr. J. Gay French.

The existence of frontal sinus suppuration in a case with polypi, after the ethmoid has been curetted, may be surmised by the appearance of pus and a small polyp at the nasal orifice of the infundibulum as well as by the discovery of pus on washing out the cavity. And it is this polypoid swelling of the infundibulum which necessitates operation upon the sinus, the object of which is to widen the fronto-nasal duct sufficiently for drainage.

Operation in chronic frontal sinus suppuration is undertaken when (1) there are indications of pus retention, whether continuous or intermittent, in the presence of pain, tenderness over the sinus, headache, sleeplessness.

(2) When, in spite of the radical removal of polypi from the nose, polypus formation continues to take place in and about the nasal orifice of the fronto-nasal duct.

(3) When, as in acute frontal sinusitis exulcerans, there is evidence in swelling of the soft parts, of extension of the disease beyond the limits of the sinus, or there is suspicion of some intracranial complication.

There are two methods of operating on the frontal sinus: the *intranasal* and the *external*. The intranasal, which is the less severe, is generally suitable for cases in the first two of the above categories—that is to say for the majority of cases —while the external is necessary in the cases of the third group.

THE INTRANASAL FRONTAL SINUS OPERATION

Description.—The enlargement of the fronto-nasal duct from the nose, so as to provide free drainage to the sinus.

Anæsthesia.—The operation can be performed under local anæsthesia, but a general anæsthetic is recommended.

Local.—Cocaine-adrenalin paste is applied to the middle meatus about the middle turbinal, the hiatus, the agger nasi, and the bulla ethmoidalis, and if a frontal sinus cannula can be passed, a few minims of sol. cocaine (10 per cent.) in an equal quantity of sol. adrenalin (1-1000) is syringed into the sinus.

General.—Chloroform preferably, as ether increases the bleeding.

The patient lies on his back with head and shoulders gently raised, the surgeon stands on the right with assistant opposite him and anæsthetist at the head. The face and nose are prepared as already detailed (see p. 226), and an X-ray picture of the sinus should be studied before beginning the operation.

Manipulation.—Under inspection, the surgeon places a middle-sized mastoid curette against the lateral wall of the nose, just external to, above, and in front of the anterior end of the middle turbinal. Firm pressure here combined with a sweep of the cutting edge of the curette downward and backward opens into the " agger cells," and leads to the fronto-ethmoidal group.

The frontal sinus cannula is passed upwards through the fronto-nasal duct into the sinus without any force or pressure, through the breach just made. After it has been passed into the sinus, it is withdrawn, and the smallest size of the Watson-Williams' frontal sinus burr or rasp passed up in its place. (See Fig. 118.) This burr, which is roughened on the anterior surface only, is made to rub down by burring the anterior bony boss or bluff formed by the floor of the sinus and the nasal process of the frontal bone. (See Fig. 119.) The burr is moved up and down, pressure being kept up in a forward direction only. The surgeon's finger placed on the thin papery bone of the inner angle of the orbit is a useful guide at this point, as the instrument can be felt through the bone, and its excursions controlled. The first burr having thus widened the infundibulum, the next size is inserted, and so on until the largest size has been used. Thereupon the duct will have been sufficiently enlarged to permit of the easy passage of the widest frontal sinus bougie of the Watson-Williams pattern.

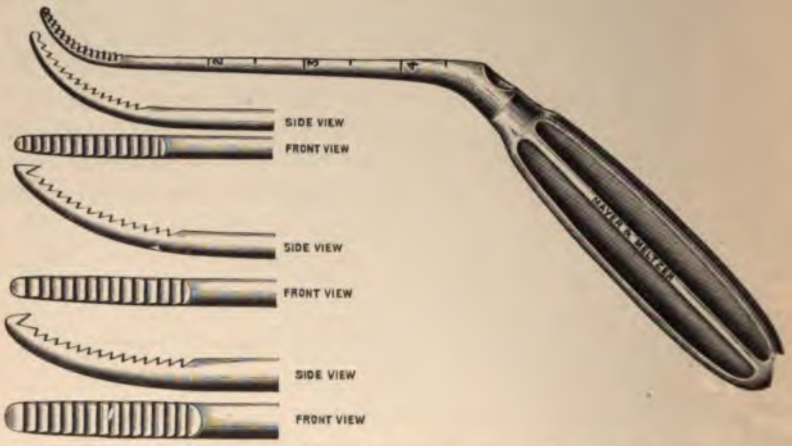


FIG. 118.—Watson-Williams' Frontal Sinus Rasps. Graduated sizes (for the intranasal frontal sinus operation).

By this process, the frontal sinus is, as it were, laid open into the nose as the duct is so much enlarged that it now forms part of a large recess continuous with the sinus.

Difficulties and Dangers.—The operation is a delicate one, unless the operator is quite at home in this region. There is a risk of going astray, and of penetrating the cribriform plate with a fatal result. The guide is the middle turbinal bone. Lateral to (outside) that bone there is safety; medial to it (to its inner side) is danger. Therefore, the middle turbinal should not be removed prior to operation if that can be avoided. Often, however, its removal has been unavoidable, or, polypoid and ethmoidal disease has already destroyed it.

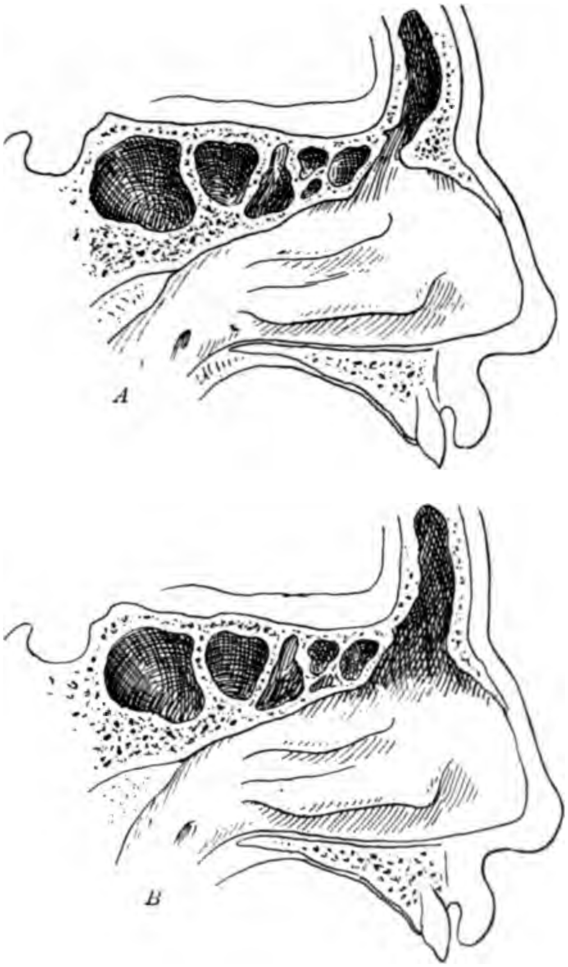


FIG. 119.—Nasal Accessory Sinuses. Intranasal operation on the frontal sinus. Widening the fronto-nasal duct (the infundibulum). After Watson-Williams.

The commonest difficulty arises from a narrow middle meatus produced by a deviated septum. In such cases, no attempt should be made to expose the infundibulum until the submucous resection of the septum is performed, and in carrying out that operation, a satisfactory access to the ethmoidal region should be secured by a sufficiently high removal of the obstructing septum. The septum operation may be undertaken at the same *séance* as the frontal sinus operation.

In any case, force must not be used. If the cannulæ, burrs, and bougies do not glide up the passage practically of their own weight, the operation should not proceed until they do. Occasionally, a little clearing of the lower end with the curette is all that is required. As a rule, the operation is rapidly and easily completed.

Sepsis.—There is a risk of septic infection in performing this operation, and experience has shown that it may induce spreading osteomyelitis (p. 372). In addition to the usual precautions, therefore, it is advisable to anoint the burrs used for rubbing down the bone with B.I.P.P.*

After-treatment.—The patient is kept in bed for several days. If there is any pain, fomentations may be applied to the forehead and face, but the after-state of the patient usually calls for no interference.

Results.—The influence of the operation on frontal sinus pain is often immediate, and for that reason, the operation constitutes the most rapid and effectual method of curing simple acute frontal sinusitis. In the ordinary chronic case also, the results of the operation are sufficiently pleasing. The sinus is now laid freely open, and it is easily washed out as often as is necessary. Discharge continues, and continues for a considerable period of time, but so long as there is an absence of other symptoms, all that is required is that the nose and the sinus should be periodically syringed out. Any mild antiseptic or sterile fluid suffices for the purpose, but after the wounds have healed a little warm alcohol, such as rectified spirit, is a useful agent.

There is a tendency, more marked in some cases than in others, for the fronto-nasal duct to re-form, and it may even become contracted and stenosed again. In that case, the operation should be repeated.

It should be clearly understood, however, that if pain is not promptly relieved by the intranasal operation, the external operation should be performed without delay.

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* B.I.P.P. is bismuth-iodoform-paraffin-paste, in the following proportions: Bismuth subnitrat., one ounce; iodoform, two ounces; liquid paraffin sufficient to make a paste. (Rutherford Morison.)

THE EXTERNAL FRONTAL SINUS OPERATION

We have already described the external operation in acute sinusitis; at the present we deal with its use in chronic frontal sinus suppuration.

Description.—The opening through the eyebrow of the frontal sinus; the clearing out of polypi and granulations from the cavity; and the freeing of the infundibulum in the nose for the drainage of the sinus.

There are two operations practised; the Ogston-Luc, and the Killian. The former is recommended for general purposes.

Indications.—1. When there are signs of pus retention in the frontal sinus, which are not relieved by the intranasal operation.

2. When there are signs of extension of the disease locally to the soft parts of the forehead or orbit.

3. When there is suspicion of intracranial complication.

N.B.—If the operation is undertaken with the sole object of curing the purulent sinus discharge, the result will often be disappointing.

A general *anæsthetic* is necessary.

The patient lies on his back with head and shoulders a little raised. The skin of the eyebrow and eyelid are carefully prepared with iodine, but the eyebrow is not shaved. A drop of sol. atropin (1 per cent.) and of castor oil should be inserted into the conjunctival sac of the eye of the same side, and the lid should be closed and covered with a pad of gauze for protection during the operation.

The surgeon stands on the side to be operated on; the anæsthetist is opposite to him; the assistant stands at the head of the table.

A skiagram of the frontal sinus should be consulted before operating. If, for any reason, a skiagram cannot be obtained, the anatomical landmark of the frontal sinus should be followed. A transverse line is drawn at a tangent to the curve of the supraorbital margin, and prolonged to intersect the middle line of the nose. At the lower angle formed by their intersection the frontal sinus will be found if it is present at all. (See Fig. 109.)

Operation.—An incision is made through the eyebrow, following its curve from its inner to near its outer end, and is carried down to the bone of the supraorbital margin, cutting through the supratrocheal, and if necessary, the supraorbital nerve and their accompanying vessels, which require catching with forceps. If the supraorbital nerve is traversed, a length of the nerve trunk should be resected. Otherwise, if the nerve is caught subsequently in scar tissue, severe neuralgia may ensue.

The pericranium having been divided and raised off the supraorbital ridge and frontal bone above it, a small frontal sinus trephine, or a gouge and mallet, is used to make the opening into the sinus. (Fig. 120.) The point selected for the opening is that indicated above. When the sinus has been opened sufficiently, pus, granulations and débris are removed, and the dimensions of the cavity are ascertained by probing.

As much of the anterior bony wall is now removed as will give satisfactory access to all the recesses of the sinus, including the orbital recess running back in the roof of the orbit. In this removal of bone, the supraorbital ridge itself is left in order to maintain the curve of the eyebrow, and no more bone of the anterior wall is removed than is necessary for purposes of exploration and cleansing.

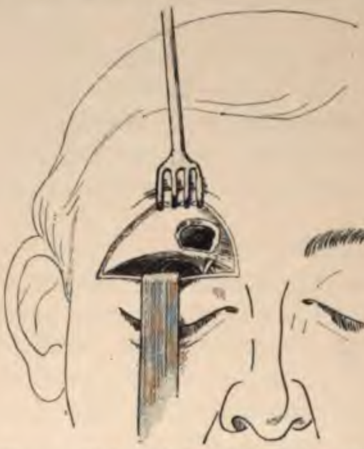

FIG. 120.—Frontal Sinus. External Operation. Opening the sinus through its anterior wall (Ogston-Luc). (After Laurens.)

Polypi, granulations and œdematous mucosa are removed with forceps, and the interior of the sinus may be cleared by being rubbed with dry gauze. Curetting the sinus wall is dangerous. The various walls of the cavity, including the intersinus septum, are inspected for carious spots or signs of disease.

Finally, the infundibulum is followed down (Fig. 121) from above into the middle meatus of the nose; its lower end is cleared of ethmoidal cells and bone; and a drainage-tube is threaded through it from above downwards to extend from the floor of the sinus as far as the vestibule of the nose.

The bone-wound and cavities are treated with B.I.P.P., the pericranium brought down over the frontal sinus opening, and the skin wound united from end to end with fine sutures.

If there has been much sepsis, or if the operation has been prolonged, it is wise to insert a tube between the lips of the skin wound into the sinus for a day while fomentations are applied.

Modifications.—The extent of the skin incision varies with the size and disposition of the sinus.

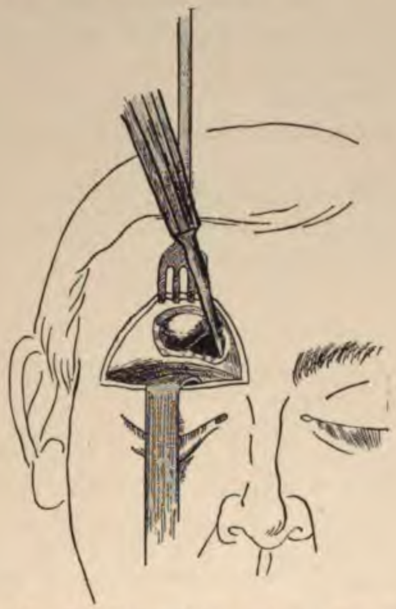

FIG. 121.—Frontal Sinus. External operation. The probe is indicated leading to the infundibulum. (After Laurens.)

Occasionally, the opening of one sinus will reveal disease in the other also If so, and it is deemed necessary to open the other sinus, this should be done through a separate incision through the other eyebrow, as an incision prolonged from one side across the middle line leaves an ugly scar.

Killian's Modification.—In this operation, the skin incision is extended down the side of the nose, and the infundibulum is opened from without through the ascending process of the superior maxilla. In addition to removing the anterior sinus wall, the inferior wall of the sinus is removed to permit of the orbital fat extending up into the sinus, in order to occupy the vacant space. A " bridge " of bone is left extending from the supraorbital margin across the gap of the infundibulum and sinus to the nasal bone, in order to maintain the contour of the orbital margin and eyebrow. The Killian operation is seldom performed nowadays.

After-Treatment.—The patient is kept in bed until the external wound is healed. Fomentations are advisable after the first twenty-four hours. Douching of the sinus is better omitted. The nasal drainage tube may be removed in from three or four days to a week. Some operators refrain from inserting any tube.

Results.—In suitable cases, where symptoms call for interference, the external frontal sinus operation is life-saving, and its performance is imperative.

It is not free from danger, as experience has shown that after this operation there is a definite risk of the onset of infective osteomyelitis.

The effect of the operation upon the suppuration in the sinus is variable. Many cases are cured entirely; many others are cured but relapse from time to time.

THE POSTERIOR GROUP

The **Posterior Group of Accessory Sinuses** comprises the posterior ethmoidal cells and the sphenoidal sinus. (Figs. 111, 112, 122.)

In many cases suppuration in the posterior group of sinuses is easily differentiated from suppuration in the anterior group, since pus from the posterior group comes from above the middle turbinal, and tends to pass backwards into the naso-pharynx, while, as we saw, pus from the anterior group appears under the middle turbinal and passes out of the front of the nose. But this clear-cut distinction is not invariable. If the situation, slope, or size of the middle turbinal is abnormal, pus from the anterior group may be directed back into the pharynx, and in the same way, pus from the posterior group sometimes finds its way forward and downward, and passes out at the anterior nares. It may, therefore, be difficult to decide by a simple inspection of the nose, whether the pus is coming from the middle or from the superior meatus. In these cases it is instructive to close up the ostia of the anterior group by a tightly-made tampon of cotton-wool stuffed into the hiatus semilunaris after all discharge has been wiped away. Having thus cut off any possible supply of pus from the anterior group, we wait for a few hours, and if, on examination, pus is then seen in the nose, it must inevitably have come from the posterior sinuses. In cases of extensive ethmoidal disease with polypoid growths filling up the superior and middle meatus, diagnostic refinements such as these are impracticable, and we endeavour to ascertain the state of the sphenoidal sinus—e.g., by catheterizing, etc. (See later, p. 365.) But it not infrequently happens that the discovery of sphenoidal sinus disease is not made until all the other accessory sinuses have been surgically dealt with.

POSTERIOR ETHMOIDAL GROUP

The posterior ethmoidal group (Figs. 111, 112, 122) comprises one or two individuals, each of which is larger than the cells of the anterior group. They border the inner wall of the orbital cavity posteriorly, and enter into relationship with the sphenoidal sinus behind. Their position and extent is variable. Occasionally a posterior ethmoidal cell comes into direct contact with the optic nerve. Suppuration in these cells is frequently combined with suppuration in the anterior ethmoidal cells, or with sphenoidal cell suppuration, and then the disease in those other cells generally dominates the clinical picture.

If the signs of suppuration in the posterior group are present, and if, on examination, the sphenoidal sinus proves to be healthy, then, by exclusion, the diagnosis of posterior ethmoidal cell suppuration may be made.

Treatment.—In such a case, with suppuration limited to one or two posterior ethmoidal cells, polypi, if present, having been removed, the cells affected may be opened up for drainage by means of Hajek's hook, or by small nasal curettes.

THE SPHENOIDAL SINUS

Anatomy.—The sphenoidal sinuses occupy the body of the sphenoid. Two in number and separated from each other by a septum, they open into the nose above the middle turbinal by an ostium situated in their anterior wall considerably above the level of their lowest recess. (Figs. 111, 112, 122.) Each of the cavities is roughly cuboid or spheroid in shape, and from its situation enters into very important relationships.

The postero-superior wall of the sinuses forms the sella turcica of the floor of the skull, and lodges the pituitary body, on either side of which is the cavernous sinus with the nerves that accompany it, the third, fourth, and sixth, and the ophthalmic branch of the fifth cranial nerve. (Fig. 189.) In relationship with the sphenoidal sinus above and to the outside is the optic nerve, which in certain cases grooves the sinus wall so deeply that the sinus may really be said to surround it.

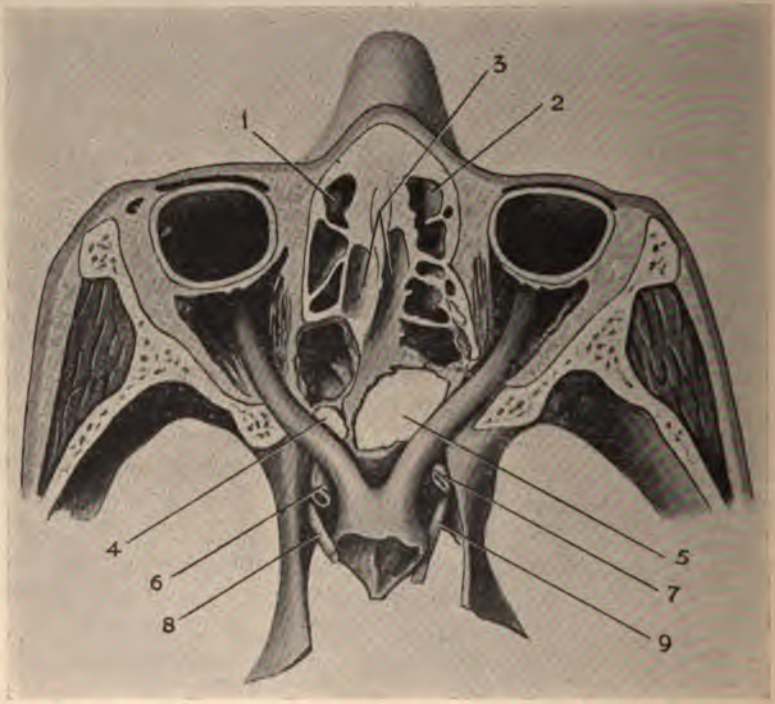

FIG. 122.—Coronal section through the face at the level of the ethmoidal cells and orbits. (After H. W. Loeb.) (1) Left frontal sinus. (2) Right frontal sinus. (3) Cribriform plate. (4) Left sphenoidal sinus (small). (5) Right sphenoidal sinus (large), in relationship with both optic nerves. (6 and 7) Internal carotid arteries. (8 and 9) oculo-motor nerves.

(Note relationship of ethmoidal cells to orbit and optic nerves.)

Anteriorly, the sphenoidal sinus is in contact with the posterior ethmoidal cells to its outer side, and with the spheno-ethmoidal recess to its inner side, into which the crescentic ostium of the sinus opens. In the middle line, more or less, the sphenoidal sinuses are in relationship the one with the other.

Like the frontal sinuses, the sphenoidal are characterized by great irregularity in size, shape, and extension. The septum is only exceptionally situated in the middle line, and the irregularity may be so extreme that the sphenoidal sinus of the one side may come into relationship with the optic nerve of the other. (See Figs. 122, 123.)

These anatomical relationships have an important influence in determining the symptoms of sphenoidal sinus suppuration.

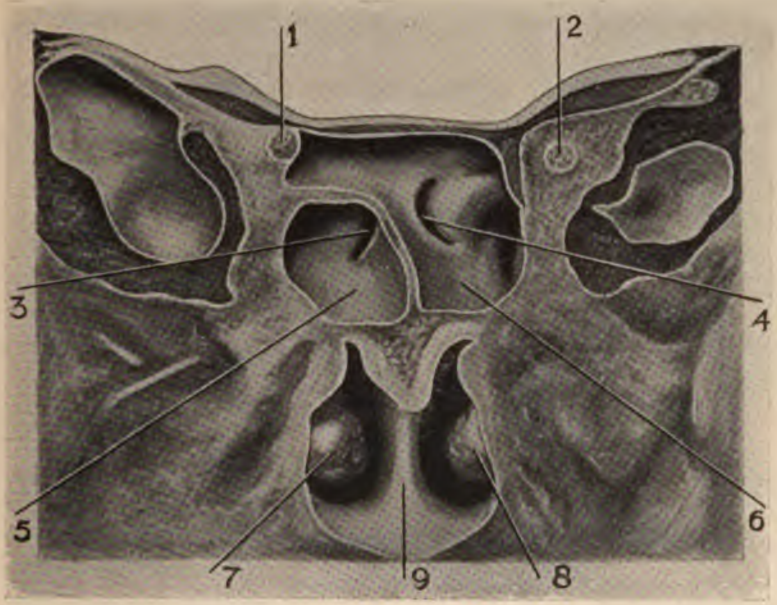

FIG. 123.—Frontal section of the face through the sphenoidal sinuses and posterior region of the orbits, viewed from behind forward. The relationship of the sphenoidal sinuses to the optic nerves is well shown. Note how the sinus of one side may extend over and come into contact with the optic nerve of the other side. (1) Left optic nerve. (2) Right optic nerve. (3) Ostium of (5) Left sphenoidal sinus. (4) Ostium of (6) Right sphenoidal sinus. (7) Left inferior turbinal. (8) Right inferior turbinal. (9) Posterior end of nasal septum. (After Onodi.)

SPHENOIDAL SINUS SUPPURATION

Suppuration in the sphenoidal sinus is probably more common than is generally supposed, for, as the sphenoidal is the most deeply situated and inaccessible of all the sinuses, the disease is frequently overlooked, and the first notice we receive of its presence may be when such serious phenomena as hemianopsia, ptosis, strabismus, or amblyopia appear. The presence, therefore, of eye symptoms indicating some interference with the optic tracts, commissure, or nerves, should always lead to a search for sphenoidal suppuration.

Symptoms.—In a typical case of sphenoidal sinusitis these are : (excluding eye symptoms, which are, as a matter of fact, unusual), purulent discharge, tending to pass into the naso-pharynx and pharynx, with the hawking and straining arising from the presence of sticky pus there, and headache.

The headache from pus-retention in the sphenoidal sinus has a definite locality ; it is felt to be at once " inside the head," and situated in an area on the vault of the cranium about half-way between the external auditory meatus and the posterior fontanelle. When intense, it is generally combined with some confusion of thought, loss of memory, or other cerebral disturbances. Headache of this character and situation, like retrobulbar neuritis of unknown origin, naturally leads to an examination of the sphenoidal sinus, and to the discovery of suppuration if it is present.

In a number of cases, however, symptoms of this class are entirely absent, and the disease is quite latent save for the discharge of pus, and even this may be so scanty and infrequent that we may never have an opportunity of seeing it as it emerges from the ostium of the sinus.

The symptoms most frequently complained of in such cases are what went at one time by the name of *post-nasal catarrh* : namely, the constant presence of a scanty, irritating, muco-purulent secretion in the naso-pharynx, with the glazed and dried mucous surface of a pharyngitis or naso-pharyngitis sicca.

In yet a third type of case, the sphenoidal sinus disease is merely part and parcel of a more or less general sinus suppuration, and the discovery of its participation is made in the long course of the examination and treatment of the multiple sinus disease.

In the inspection of the nose, pus from the sphenoidal sinus appears first in the spheno-ethmoidal recess, where it may await discovery on examination with the nasal endoscope. At a later stage, and when plentiful, it will be seen in the post-nasal mirror oozing out from above an œdematous middle or superior turbinal.

Neither transillumination nor examination by X-rays is of any service in the diagnosis of sphenoidal sinusitis ; we are, therefore, compelled to rely upon the results of catheterization, either through the natural ostium or through a puncture made in the wall of the cavity.

The sphenoidal sinus cannula is a silver tube about 13 centimetres long, slightly curved at the point, and graduated up to 10 centimetres. (Fig. 124.)

In order to pass this cannula, we begin by insinuating the tip between the middle turbinal and the septum, crossing the former just behind its middle point. Having reached the superior meatus, the point of the instrument is then directed backwards and upwards, in a line towards the posterior fontanelle, until the roof of the nose is reached. Then, by turning the point somewhat upwards and outwards, and trying gently here and there to push it further in, the ostium of the sinus is generally entered. The cannula, in contact with the roof of the nose, enters to about a depth of 7 to 8 centimetres, and when it enters the sphenoidal sinus, it passes onward for 1 or 2 centimetres more. When it is right inside the sinus, attempts to depress the point in a direction downwards and backwards will fail. A further indication that the instrument is in the sphenoidal sinus is obtained when the point of the catheter is made to impinge upon the posterior wall of the sinus, that is on the body of the sphenoid, as there is felt, on tapping that bone, a peculiar sense of solidity, as if the cannula were striking dense, smooth bone, bare, or almost bare, of any mucous covering. The sensation is a peculiar one, and quite unmistakable.

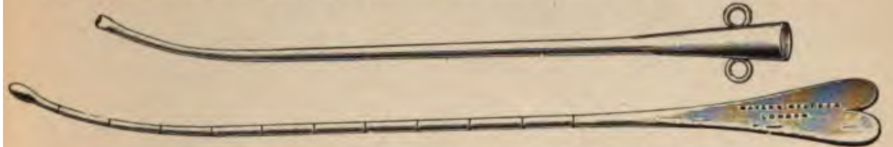

FIG. 124.—The Sphenoidal Sinus Cannula and Probe.

In order to pass the catheter, it may be necessary to remove the middle turbinal. After this has been done, one can often inspect the ostium of the sinus, and perhaps see pus welling out of it.

When the catheter has entered the sinus, the character of the contents of the cavity may be discovered by blowing through the tube and watching the secretion as it trickles down the posterior pharyngeal wall. If the quantity of pus is small, we may aspirate some of it into the catheter, and examine the contents of the instrument after withdrawal. If pus has been found, as in the case of the frontal sinus, a stream of warm boric solution is passed through the cavity; but if no pus is discovered, douching should not be practised.

In cases where the catheter cannot be made to enter the natural ostium, it is advisable to puncture the sinus with a long trocar and cannula. In that case a Watson-Williams' sinus syringe, attached to a long, blunt, straight sphenoidal trocar and cannula is passed backwards and upwards in the direction indicated above for the passage of the catheter. When the body of the sphenoid is reached, no attempt is made to find the natural ostium, but by pressure, the blunt point of the instrument is driven directly through the anterior bony wall into the sinus cavity. A small quantity of sterile saline is then injected, aspirated back into the syringe again, and examined (Watson-Williams).

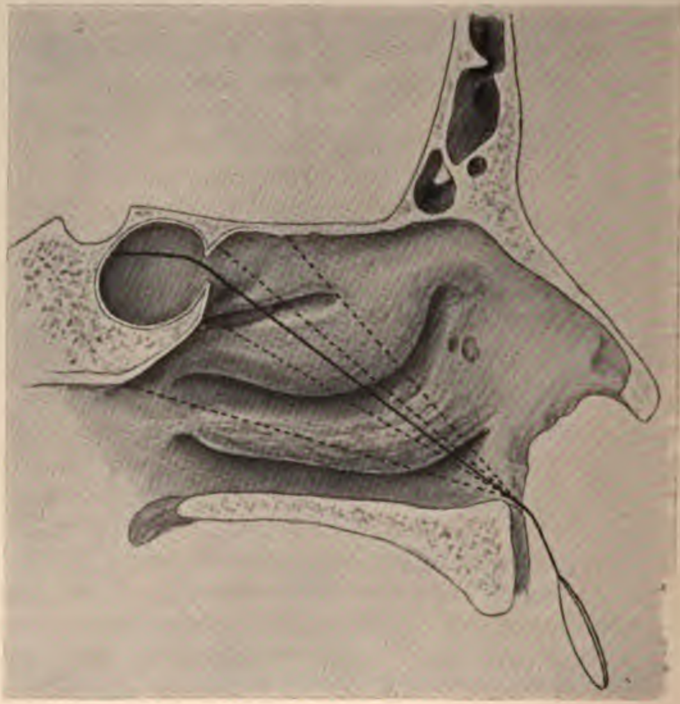

FIG. 125.—Probing the Sphenoidal Sinus. (After Hajek.)

Prognosis.—In spite of the depth and apparent inaccessibility of the sphenoidal sinus the prognosis of the sphenoidal sinusitis under treatment is favourable. If free drainage can be provided and maintained, recovery will follow after a time. The recovery affects also any changes that may be present, provided that they have not progressed too far. Thus, if optic atrophy has set in, no operation, however successful, will ever restore the vision.

Treatment.—The indication is to open and drain the sinus by an enlargement of its natural ostium. The operation must be preceded by an exposure of part of the anterior wall of the sinus either by removal of polypi and polypus-bearing ethmoidal bone or by middle turbinectomy. These regions of the nose are very vascular, and the operation can therefore only be carried out under the guidance of the sense of touch. The area should be treated before operation with cocaine-adrenalin paste, care being taken not to let the anæsthetic find its way into the pharynx.

SPHENOIDAL SINUS OPERATION

The operation can be performed under local anæsthesia, but most British operators prefer also a general anæsthetic.

The patient is laid on his back, with head and shoulders slightly raised. When the bleeding becomes profuse, the face should be turned on one side, and if necessary, a sponge may be held in the naso-pharynx. (See also p. 278.) Some operators prefer a captive sponge in the naso-pharynx.

Operation.—Under guidance of head-light or head-mirror, and using the long-bladed nasal speculum, it is often possible to see the ostium, and to pass the first cutting instrument, which is Hajek's hook, under inspection. If not, the sphenoidal sinus cannula is inserted, its direction, route, and depth noted, then it is withdrawn, and the Hajek hook inserted in its place, and to the same depth.

The hook, as it lies in the sinus, is turned downwards, and then pulled suddenly and forcibly out of the cavity. This is repeated two or three times with some slight alteration in the disposition of the hook. (Fig. 126.)

Next, the hook is passed into the cavity and its point is turned directly outwards so as to tear through the anterior wall of the sinus lateral to the ostium, a step which often opens up the adjoining posterior ethmoidal cells also.

Into the ostium thus enlarged biting sphenoidal sinus bone forceps are passed in such a way as to grasp the same lower lip, one blade being within the sinus, and one without, and they are closed strongly, with the object of removing the thick wedge-shaped lower lip of the ostium. This last is the important step in the operation, and is frequently very hard to accomplish.

On no account should the sphenoidal sinus be curetted, as the optic nerve may be traversing the cavity without any bony covering. For the same reason, the operator is careful not to deviate more than a few millimetres laterally from the spheno-ethmoidal recess and the ostium sphenoidale.

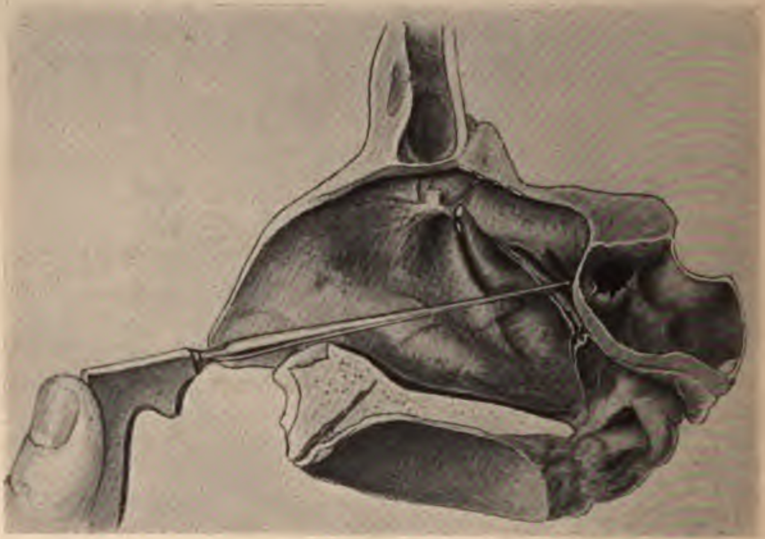

FIG. 126.—Enlargement of the ostium of the sphenoidal sinus with Hajek's hook. The middle turbinal has been removed. The hiatus semilunaris is visible, and so also are openings of posterior ethmoidal cells. (After Hajek.)

Hæmorrhage is usually profuse for the moment, but soon ceases. Occasionally, a wound of the sphenoidal artery gives rise to serious bleeding, and necessitates a deep and firm packing with ribbon-gauze. Packing, however, should if possible be avoided, even at the expense of considerable loss of blood, as the effects of imprisoning septic material within the sphenoidal region are sometimes serious, meningitis or cavernous sinus thrombosis being induced.

Difficulties and Dangers.—The first and chief difficulty is to secure an entrance to the sinus. If this can be accomplished, the operation presents no difficulties. But sometimes both tact and patience are required.

The use of a long gouge and mallet in this region is not recommended, as the body of the sphenoid—the posterior wall of the sinus—is sometimes quite thin and may easily be broken through.

Results.—The results of the operation are good. The wound soon heals up, and although the enlarged ostium tends to contract somewhat, the opening left is usually sufficient both for drainage and for lavage, if that is required.

It is important to ensure at the operation as thorough a drainage of the adjoining ethmoidal cells as can be obtained.

MULTIPLE SINUSITIS AND PANSINUSITIS

We have constantly alluded, in dealing with individual sinuses, to the association of one or more of the group-members in disease, and it only remains for us to consider the association a little more in detail, especially from the point of view of treatment.

Multiple sinusitis is the rule. Sinusitis limited to one sinus only is the exception. Sinuses which adjoin are, naturally, more often associated in disease than sinuses, such as the frontal and the sphenoidal, which are distinct and separate from each other.

In the same way, the members of the groups, anterior and posterior, tend to infect each other, or to be infected together simultaneously. Thus, one habitually in examination pays closest attention to the members of one group that seems to be affected.

The ethmoidal cells, holding, as they do, the central position, constitute the bridge from one group to the other, so that when both groups are involved (*pansinusitis*) the ethmoidal cells are also diseased, and generally are occupied by polypi in large numbers.

Clinically, the usual history of such a case is that it comes for relief from nasal obstruction. This, on examination, is found to be due to polypi blocking the nose, and arising as a consequence of ethmoidal suppuration. When these are removed, and the ethmoidal region curetted, further examination reveals suppuration in antrum or frontal sinus or sphenoidal sinus, or any combination of these.

It is remarkable that the mechanical obstruction to the nasal breathing may be the only symptom observed by the patient even in extensive and long-continued sinus suppuration.

The *Treatment* of multiple and pansinusitis is often a prolonged and tedious affair. It is seldom possible to effect a clearance of the ethmoidal disease and to open up the other sinuses in one operation, and the possibility is still more remote if there is also a deflected septum to be rectified before proceeding to the deeper sinuses. The procedure usually adopted is somewhat as follows: first of all, the nose is cleared of polypi, and perhaps curetted; next, the septum is straightened, and perhaps nasal antrostomy, or the intranasal frontal sinus operation performed. Later, the sphenoidal may be opened, and the ethmoid may require a second curetting.

This course of surgical intervention may take from three to six months or longer to be completed, and even after all is finished and done with, the discharge of pus from the nose will continue remittently for an indefinite period of time.

If, however, there are no signs of pus retention, and if polypus formation comes to a standstill, all that is necessary is for the nose and its cavities to be douched periodically with some non-irritating sterile or mildly antiseptic solution.

During this period, the patient should be kept under observation, the nose being carefully examined at short intervals. It is by the presence or absence of local œdema or polypus formation rather than by the presence of pus that we estimate progress. If œdema of the mucosa in the ethmoidal region or elsewhere persists or recurs, with or without polypus formation, we regard the disease as still active and the cells in need of drainage. If, on the other hand, the œdema and swelling diminish and finally pass away, the underlying bony structures becoming prominent, the disease may be considered to be getting well.

If signs indicative of continued disease arise or persist, then, by the timely nibbling away of some polypoid bud, or other minor manipulation, we are able to forestall and to prevent any serious developments, and so gradually to shepherd the case into safety.

Syringing and douching the nose ought to be carefully regulated. As soon as the discharge ceases to be plentiful, the nasal syringing should be reduced, and as soon as possible it should be stopped.

Rhinitis sicca is the final stage of many of these cases, and the most successful rhinologist is he who can accomplish the cure of nasal sinus suppuration with the least sacrifice of the physiological activity of the nose, but the conservative surgeon will find his efforts strongly supported by Nature if he preserves the spongy turbinal tissue intact.

Vaccine Treatment.—The treatment of nasal sinus suppuration by vaccines will be either harmful or beneficial according to the stage in which it is employed.

While sinuses are in the stage of active disease, before they have been cleared and laid open for drainage and aëration, I have seen vaccines do harm: increasing the discharge, and livening up the local inflammatory reaction.

But after all the diseased sinuses have been operated on, and the patient has entered upon the convalescent stage; that stage may be shortened by the administration of autogenous vaccines, prepared from the pus lying in the sinus. My practice is to give the vaccine in two or three courses of about six weeks or two months each with an interval of about six months.

W. D. Harmer reports good results from vaccines of streptococcus and Friedländer bacillus. In acute streptococcus infection, the vaccine treatment was often remarkably successful.

In any case, once the sinuses are opened sufficiently, and kept open, we may prophesy cure. As time passes, the purulent content of the discharge will gradually diminish, and as it does so, the quantity of the discharge will also grow less, until finally, it dries up for good. This process may be extremely tardy, lasting, it may be, for from three to five years, and it is generally subject to interruption from time to time; but, provided always that the sinuses can be kept open and that polypus-bearing areas can be eradicated, the ultimate destiny of these cases is recovery. In making this statement, we assume, of course, that the patient has not been the victim of one of the graver complications of sinus disease during the period of active sinusitis.

COMPLICATIONS AND SEQUELÆ OF ACCESSORY NASAL SINUS SUPPURATION

We have already, in passing, considered the subject of the *oculo-orbital complications* of sinus suppuration, but it is also necessary to add that certain septic diseases of the globe, such as irido-cyclitis, may be set up, it would seem, by the presence of purulent disease in the maxillary antrum, and others of the accessory sinuses. In such diseases of the eyeball, therefore, as well as in the more serious retrobulbar neuritis, the sinuses should be systematically surveyed, and if any purulent disease is found, the affected sinus should be freely opened up by operation.

In examining the sinuses in such cases, as well as in cases in which the presence of " neuralgia " or " headache " raises the question of nasal sinus suppuration, it is necessary to state that *the absence of any sign of disease inside the nose is not sufficient to exclude sinusitis from the diagnosis.*

All cases must also be submitted to X-ray examination by a radiologist experienced in the skiagraphy of the nasal sinuses, as by this means the presence of pus in one or other of the sinuses may be unexpectedly revealed. But even X-ray examination is not infallible, so that in the presence of serious orbito-ocular trouble, such as optic neuritis, for example, the only satisfactory method of excluding suppuration of the sphenoidal and posterior ethmoidal cells—the sinuses most likely to set up eye disturbances—is to operate and open them up.

In view of the irregular anatomical relationships of the sphenoidal sinus, if operation is undertaken for oculo-orbital disease, the sphenoidal sinuses of *both* sides should be opened up, even when the eye symptoms are only unilateral. (See Figs. 122, 123.)

The subject of *septic meningitis* from nasal infection is discussed at p. 553, and that of *frontal lobe abscess* at p. 348.

DIFFUSE CRANIAL OSTEOMYELITIS

This is an infective disease of the bone, starting in an infected sinus, and tending to spread and involve the entire cranium.

Etiology.—The precise factors which lead to the onset of the disease are unknown, but experience has taught us that it follows frontal sinusitis more frequently than sinusitis of any of the other cavities, and that extensive operative exposure, and, especially, vigorous curetting of the walls of the sinus, predispose to its occurrence.

Pathology.—The bone attacked by septic organisms dies *en masse*, but the soft structures retain their vitality, and the consequent reaction, local and general, to the presence of the dead bone and septic organisms, is responsible for the clinical phenomena of the disease.

The extension of the disease is alternately sluggish, and by leaps and bounds, unaccountable pauses occurring in its progress, because, in spite of the continuity of the bone of the cranial vault, only circumscribed areas of the bone are attacked and devitalized at one step.

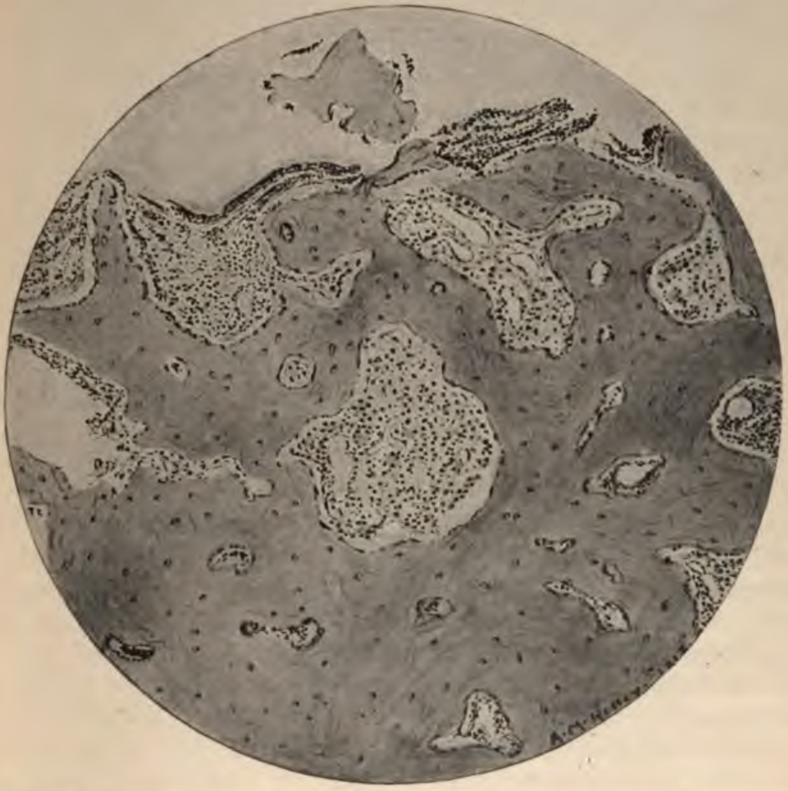

FIG. 127.—Osteomyelitis of Frontal Bone. Osseous tissue invaded by granulation tissue.

The bone affected consists, however, of the entire thickness of the vault, outer table, diploë and inner table. The diploë is filled with pus; abscesses form deep to the inner table—extradural abscess—and superficial to the outer table—abscess of the scalp; and as time passes granulation tissue, forming in and around the necrosed areas, invades (Fig. 127) and erodes the plaques of necrosed bone, which come to bear a worm-eaten and riddled aspect.

The proximity of this septic bone-disease to the intracranial structures frequently entails the development of septic meningitis or brain abscess, and may lead to death.

Symptoms and Progress.—The disease may arise spontaneously from (a) acute sinusitis exulcerans; (b) in the course of a chronic sinusitis; or (c) it may appear during convalescence from an operation on the sinus.

The onset is quiet and insidious, with the appearance of a small, pale, puffy swelling of the soft parts—usually the forehead, in the case of the frontal sinus; the side of the nose, in the case of the ethmoids; and the cheek in the case of the antrum. The swelling may be tender, and pain is sometimes a prominent feature in the case. At the same time, the temperature rises to a subfebrile reading.

Incision, or the breaking of the little abscess, reveals a patch of dead bone, and in the course of a few days, one or two more subcutaneous swellings develop a little further afield, each marking a new extension of the disease.

If the disease is allowed to proceed, these abscesses discharge pus for a time and then subside, and the pain, swelling, and fever come gradually to a standstill.

Then, in the course of a few weeks, a further recrudescence appears with the signs of extension of the bone disease in a similar crop of quiet abscesses, with swelling, pain and fever; and so on indefinitely, until the supervention of an intracranial complication, or the slow effects of the drain upon the strength by pain, fever, and discharge, lead to the fatal issue.

Prognosis.—Osteomyelitis arising spontaneously can be arrested, often permanently, by radical removal of the diseased bone.

Cases occur of spontaneous recovery after the exfoliation of a sequestrum in osteomyelitis secondary to maxillary antrum suppuration limited to the superior maxilla, but when the disease attacks the vault of the cranium, it seems to be invariably fatal unless arrested by operation.

The post-operative type of the disease would seem to be seldom curable, even when it is radically treated.

Treatment.—As soon as the diagnosis is made, the whole of the diseased area of the bone must be completely exposed and entirely removed. This may entail extensive incisions, and the removal of wide areas of bone. But the earlier the intervention is made, the less, naturally, will be the necessary sacrifice. Both tables of the skull must be removed, the dura being laid completely bare, and the nibbling away of the bone—best done with cutting bone-forceps—is carried on until the whole of the dead bone is removed. When the healthy zone is reached, freshly sterilized instruments should be employed.

Finally, the cut edge of the bone is rubbed liberally with B.I.P.P.

When the disease involves the flat bones of the face, such as the nasal bones, and also when it has destroyed the supra-orbital ridge, the necessary removal of the bone entails the infliction of great disfigurement.

When the frontal eminences only are sacrificed, however, the bone will be restored if the patient survives.

The effect of spreading osteomyelitis of the posterior ethmoidal and sphenoidal region is not clear, but it is probably responsible for the rare and unexpected deaths from meningitis and cavernous sinus thrombosis, respectively, that sometimes occur in the course of ethmoidal and sphenoidal sinusitis.

In addition to these the rarer and more serious sequelæ of nasal sinus suppuration, we have also such simpler consequences as chronic pharyngitis, " post-nasal " catarrh, and the chronic laryngitis.

With regard to the last of these, which is not very common, it is generally supposed that the irritant causing the disease is pus, which finds its way from the nose to the larynx. That pus does reach the larynx from the nose is, however, very doubtful.

Patients with nasal sinus suppuration, especially when the maxillary antrum is involved, frequently suffer from chronic cough with copious purulent expectoration, probably arising from *septic bronchial catarrh*, as the lungs show no sign of cavitation, nor is any indication of tuberculosis visible on X-ray examination. The sputum, moreover, does not contain tubercle bacilli. This condition seems to get well slowly after the maxillary antra and the other diseased sinuses are opened and drained.

Rhinomycosis.—(Fungus Infection of the Nasal Cavity and Accessory Sinuses.)

W. D. Harmer, H. Tilley, W. B. Brownlie, and others, have recently drawn attention to this condition, and personal experience would suggest that the disease is by no means a rarity. It has been mistaken in the past for purulent sinusitis.

The infections so far recognized have been the aspergillus and the *oidium albicans* (the thrush organism). Whether the disease is primarily due to the mould infection, or whether that infection is merely secondary to a chronic septic process is not yet definitely proved. But the probability is that the mould infection is primary.

Pathology.—The disease affects women for the most part; it seems to occur in members of the same family and to affect farm and field workers.

In the maxillary antrum the material consists of a mucinoid matrix (probably inspissated mucin) interpenetrated by elongated groups of polymorphonuclear leukocytes, and simulating microscopically a new growth of an endotheliomatous character. The mycelium can be demonstrated in the mucinous matrix (S. G. Shattock, *Journ. of Laryng.*, vol. xxx., p. 145).

The *symptoms* are characteristic. There is nasal obstruction with sneezing and a mucous or muco-purulent discharge containing "small masses of a whitish-grey, semi-translucent, viscous material" (H. Tilley). Neuralgic pains in the face and cheek are complained of.

The nasal mucosa is œdematous and does not shrink on cocaine being applied. The ethmoidal regions contain polypi, round, small and multiple, the removal of which is attended with very little bleeding.

Transillumination shows the antra to be dull, but on proof-puncture, either the cavities cannot be washed through at all, or else the fluid returns clear. Sometimes, however, prolonged lavage results in the expulsion of a quantity of mucoid material.

The disease leads to the atrophy, and finally to the disappearance of the bone of the nasal wall of the antrum.

When that cavity is opened, it is seen to contain a bluish-grey glistening material, which, on removal, resembles in colour and consistency "the sticky contents of a muscatel raisin." (H. Tilley.)

The disease may affect the antrum, the ethmoidal region, the lacrymal apparatus, the naso-pharynx, and perhaps the sphenoidal sinus.

Treatment.—If the operative opening up of the affected sinuses and the removal of the morbid material fails to cure the disease, the patient should be given one of the iodide salts internally. Harmer obtained cure by administering gr. xx. sod. iodid. three times a day. (*Journ. of Laryng.*, vol. xxviii., p. 494.)

For *Mucocele* of the sinuses, see p. 293.
For *Malignant Disease*, see p. 294.

CHAPTER X

AFFECTIONS OF THE NASO-PHARYNX

At birth, the naso-pharynx is represented by a small, flat space between the soft palate and the posterior pharyngeal wall, which will scarcely, if at all, admit the point of the little finger. The posterior edge of the nasal septum, which in adult life is vertical, is in infancy nearly horizontal. This arrangement holds with but little alteration until the child reaches the age of two or three years. After that date, the naso-pharyngeal cavity begins to enlarge in all directions, until the adult dimensions are reached about the age of twenty-one.

Two modifications in this development are met with : first, that of defective expansion, when the adult naso-pharynx is small ; and secondly, that of excessive size.

Defective expansion.—Smallness of the naso-pharynx is found as part and parcel of the imperfect development of the upper passages in cases of neglected adenoids where the narrow nostril, Gothic palate, and rodent face are present. It also occurs in consequence of the early synostosis of the cranial base in cretinism.

Another cause of defective space in the naso-pharynx is found in excessive development and undue prominence of the " Eustachian cushions " of cartilage that surround the orifice of the Eustachian tube.

Finally, the cavity may be encroached upon from behind by what was at one time believed to be an enlarged tubercle of the atlas vertebra, but has more recently been attributed to displacement by lateral rotation of the upper cervical vertebræ (E. F. Cyriax).

Enlargement of the naso-pharynx is met with as a consequence of congenital shortness of the hard palate. (See also p. 25.)

ADENOIDS

The Pharyngeal Tonsil, a collection of lymph follicles on the roof and posterior wall of the naso-pharynx, is liable to the same diseases as the other tonsils. The most usual abnormality is hypertrophy, commonly known as post-nasal adenoids, or simply "adenoids."

The naso-pharyngeal tonsil, normally present at birth, should undergo atrophy about puberty, and disappear before the age of twenty. An abnormally large naso-pharyngeal tonsil (adenoids) gives rise to symptoms such as obstruction to nose-breathing, deafness, suppuration of the middle ear, etc. These generally appear some time between the ages of five and puberty, but they may give rise to symptoms in early infancy, and may linger on into old age.

It will be found that upwards of 30 per cent. of all children in Britain suffer from adenoids, and require operation.

The cause of adenoids, like the cause of hypertrophy of the faucial tonsils, is unknown, but modern opinion tends to the belief that the hypertrophy of this "ring" of lymphoid tissue, known sometimes as *Waldeyer's ring*, and including the naso-pharyngeal, the faucial and the lingual tonsils, is a reaction to septic infection of the nose and naso-pharynx. As we have remarked elsewhere, it is a fact that adenoid symptoms, and so, presumably, the adenoid hypertrophy, often date from one of the exanthemata, such as whooping-cough or measles, in which nasal "catarrh" is a prominent symptom; and, moreover, since in nearly all cases of hypertrophied faucial tonsils in childhood, adenoids exist as well; while, on the other hand, many children are found suffering from adenoids in whom there is no hypertrophy of the faucial tonsil, we may presume that the infection passes first to the naso-pharyngeal tonsil, and secondly, to the faucial tonsil.

Another circumstance pointing to bacterial infection as the cause of the disease is the fact that these lymphoid masses, when hypertrophied, are very susceptible of infection, and seem to grow larger for a time in consequence thereof. In later years, however, the same cause brings about a shrinking of their mass by the production of fibrosis and cicatricial contraction, so that adenoids which in childhood are soft, friable, and deeply cleft by sulci, become, in adult life, hard, tough, and smooth on the surface. (Fig. 128.)

Adenoid vegetations spring from the roof, posterior wall, and to some extent the lateral walls, of the naso-pharynx.

The *Symptoms* they induce are secondary to (1) the liability of the adenoids to infection or "catarrh"; (2) the obstruction they present to nasal respiration; (3) Eustachian obstruction.

1. *Symptoms due to* infection of the adenoids; or *adenoiditis*. This, perhaps, is the most frequent and most constant of all the many symptoms that may be induced by the presence of adenoids. Translated into homely phraseology, it amounts to this, that the patient is always "catching cold," the fact being that the local resistance of the immature lymphoid tissue to the ordinary bacteria of sepsis is very low, and, as a consequence, it is continually being subjected to the inflammatory reaction ("catarrh") in order to overcome bacterial invasion.

The catarrhal infection, whatever its nature may be, radiates from this breeding place in the naso-pharynx to the nose—a "running nose" in a child is another sign of adenoids;—to the larynx, inducing spasm of the glottis, "croup," and hoarseness; to the trachea and bronchi, causing bronchitis, and possibly asthma, and leading, along with the nasal obstruction, to the development of round shoulders, flat chest, and recession of the lower costal cartilages. Finally, the catarrhal inflammation may spread to the ears (see below).

FIG. 128.—*A*. Soft adenoids of childhood showing longitudinal clefts. *B*. Hard adenoids of adult life showing smooth surface left after obliteration of the clefts (natural size).

The constant succession of "colds" is accompanied with many of the signs of general toxæmia in chronic anæmia and malnutrition, together with heaviness and sluggishness of the mind, and backwardness in bodily and mental development. Sometimes these constitutional phenomena are attributed to the effect of the nasal obstruction, and no doubt correctly so, but to a great extent also they may be explained by the general poisoning of these children, seeing that although it is true that nasal obstruction and partial deafness aggravate these symptoms, they are also noticeable in children who have neither nasal obstruction nor loss of hearing.

2. *Symptoms due to respiratory obstruction.*—Many, but not all children with adenoids are mouth-breathers, snoring when asleep, and making a snuffling and choking noise when eating and drinking. As they must breathe through the mouth and

chew simultaneously, they can never "eat with the mouth closed." At night there may be night-terrors, spasm of the glottis, nocturnal enuresis, and other molestations by the Powers of Darkness. The naso-pharyngeal obstruction renders the voice toneless and flat. The child has a low bridge to the nose; the eyelids are puffy and swollen; and the facial expression is stupid and vacant.

It is to be noted that this picture only applies to cases in which the adenoids have been present for some considerable time.

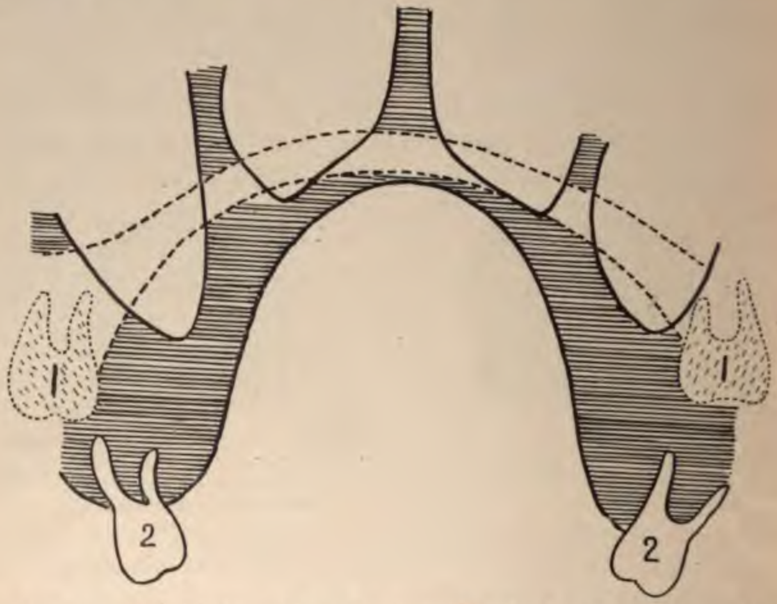

FIG. 129.—The Gothic palate, due to neglected adenoids (diagrammatic). (1) The normal palate and alveolar process. (2) The deformed palate and alveolar process (Lambert Lack).

It is the fully developed adenoid syndrome, but it should never be allowed to get as far as that point, and to be quite truthful, it seldom does nowadays. The adenoids are usually removed long before this period.

If, however, the early symptoms are ignored and the case is neglected, serious secondary results are almost inevitable: the nose and naso-pharynx fail to undergo their full normal development; the upper jaw becomes narrow (Fig. 129); the nose is pinched; the teeth crowded; the incisors protruding; the bite open (see also p. 13); and we have before us "the rodent face," which is the typical English face of the Continental caricaturists.

3. *Symptoms due to Eustachian obstruction*, obstructive deafness, that is to say, with indrawing of the tympanic membrane—are the effect of Eustachian catarrh secondary to the turgid and swollen condition of the Eustachian lips and walls, when adenoids are present. During the periodic attacks of adenoiditis, these aural symptoms become more noticeable, and attract, by the deafness they induce, the attention of parents and school-teachers. A number of the children also suffer on these occasions from fugitive attacks of acute catarrh of the middle ear with sharp earache, generally subsiding without perforation of the membrane, but sometimes leading to otorrhœa of short or long duration. Suppuration of the middle ear in children is due to adenoids in the large majority of cases; the minority being due to tuberculosis. (See also p. 517.)

It is perhaps the case also that the chronic catarrhal deafness of middle life is due to late secondary effects of these transitory attacks of non-discharging, acute catarrh of the middle ear in childhood.

In any particular case, let us say in conclusion, any of the above symptoms may be present in combination, but it is also necessary to add that only one of the groups may be present, and that in quite a minor degree. One frequently finds parents sceptical of the presence of adenoids because the child does not snore. But, as we have already said, snoring is one of the later phenomena of the disease.

In other words, the presence of adenoids in any case, whether in child or adult, can only be excluded by examination.

Examination.—Posterior Rhinoscopy.—Adenoids show as dark-red masses, encroaching on the upper half of the nasal septum in the image. It must be remembered that we see these growths very much foreshortened. The vertical sulci between the several masses are often visible, and serve to distinguish adenoid vegetations from every other nasal or naso-pharyngeal growth. In early childhood, as a rule, the sulci are well marked, but in later childhood, youth, and adult life, they tend to become obliterated. (Fig. 128.) Thus the adenoid masses become smoother, as well as harder, with age. At this time their partial atrophy may permit the view of the recessus medius, or pouch of Rathke, a median cul-de-sac of fœtal origin.

Examination with the finger will result in the palpation of the soft, movable, corrugated masses of adenoids. The "feel" of the sulci renders the diagnosis sure.

Diagnosis.—If examination with the mirror or finger be carefully carried out, adenoids, when present, will never be missed.

At the same time, a certain experience in digital examination of the naso-pharynx is necessary before one can be quite confident as to what one actually feels. The examiner should never establish his diagnosis of adenoids on the presence of blood on the examining finger. **Naso-pharyngeal palpation should never cause bleeding** ; if it does so, undue roughness has been used, and roughness will cause a perfectly normal naso-pharynx to bleed.

The diagnosis between adenoids and other nasal and naso-pharyngeal growths, such as mucous polypi, fibroma, retro-pharyngeal abscess, etc., depends upon the fact that in these other tumours, the surface of the swelling is quite smooth, and that their attachments are rarely quite the same as those of adenoid vegetations, which, as we have already noted, cluster round the posterior wall and roof of the naso-pharynx.

Prominent bony processes, such as the atlas tubercle, the hamular process, or process of the palate bone, may possibly be mistaken for adenoids by careless or hurried examiners.

Hypertrophy of the posterior lip or cushion of the Eustachian orifice may also be mistaken for adenoids, but its smooth cartilaginous "feel," and bilateral position should prevent such an error being made.

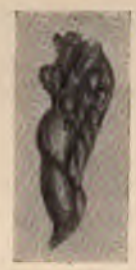

FIG. 130.—Ringlet of adenoids revealed by the endoscope, and removed from the fossa of Rosenmüller of an adult (slightly enlarged).

In adults with symptoms of exudative catarrh of the Eustachian tube or middle ear, the presence of adenoids may be limited only to a small ringlet filling up the fossa of Rosenmüller, which lies behind the posterior lip of the orifice of the Eustachian tube. (Fig. 130.) They are easily missed on digital examination, and may only be discovered by endoscopic inspection. Their discovery and removal may be of the first importance in the treatment of the ear disease and its consequent deafness.

Prognosis.—The prognosis, as regards the adenoid masses themselves, is good. If left alone, they become less and less as the years go on, until at about the age of twenty they entirely disappear.

But their works endure.

If not removed promptly when first discovered—if not removed also each time they recur, the unfortunate patient will very probably be launched into adult life burdened with defects which will seriously handicap his future.

On the other hand, the complete removal of adenoids is reflected almost immediately in a striking improvement in all the symptoms, and if the removal is effected early, the symptoms entirely disappear.

With regard to recurrence after operation, parents should be warned that recurrence is not uncommon in children operated on before the age of seven years. After that age, recurrence is much less frequent. And while recurrence is more likely to occur when the growths have been incompletely removed, it may, nevertheless, surprise us even after the most radical extirpation. I have myself removed adenoids as often as three times in the same patient.

Nevertheless, as we have already said, they ought to be removed as often as they recur.

While the above is true, and must be stated with regard to recurrence, on the other hand, it may also be claimed that recurrence is, in point of fact, very exceptional at any age when the operation is efficiently performed.

The *Treatment* of adenoids is removal by operation.

THE OPERATION FOR ADENOIDS

Contra-Indications.—The operation should be postponed if the child is suffering from any intercurrent malady, or has a temperature. Adenoids should not be removed during the acute stage of middle ear suppuration.

It is the rule not to remove adenoids in cleft-palate or in atrophic rhinitis.

The *Preparation* is the same as for the removal of tonsils, as the two operations are frequently performed at the same time. The same directions as to position, anæsthetic, and so on, apply to both operations, for which, see p. 52.

Operation.—When both tonsils and adenoids are to be removed, the adenoid operation follows the tonsil operation.

1. **Removal by the La Force Adenotome.** (See Fig. 131.)—The instrument should be tested before insertion, as the flexible blade is liable to buckle, and may break while in the mouth.

Under inspection, the adenotome is passed open, into the naso-pharynx, and is held precisely in the middle line. It is then pressed back to bring it as close to the posterior pharyngeal wall as possible, and, with the finger supporting its upper surface as it moves, the sliding blade is pushed home. The instrument is then removed from the mouth still closed, and opened after removal, the adenoid mass being shaken out of the receptacle.

Again it is inserted, this time being turned a little obliquely towards one side of the naso-pharynx, in order to remove the lateral clusters that may have escaped the first shaving. And again the instrument is removed closed, and the adenoids are shaken out.

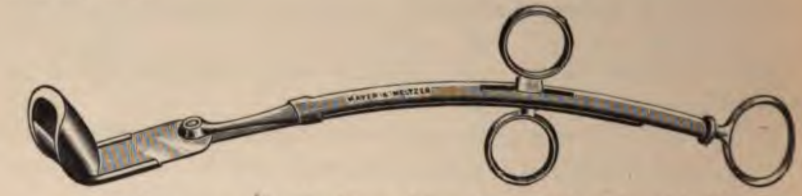

FIG. 131.—The La Force Adenotome (with sliding blade).

This is repeated a third time, to remove the lateral cluster of the other side.

Even then, some remnants may still remain attached to the lateral wall, but they can be easily extirpated with a few strokes of a simple adenoid curette. (Fig. 133.)

To make certain that all the adenoids have been removed, it may be advisable to pass the finger into the naso-pharynx and

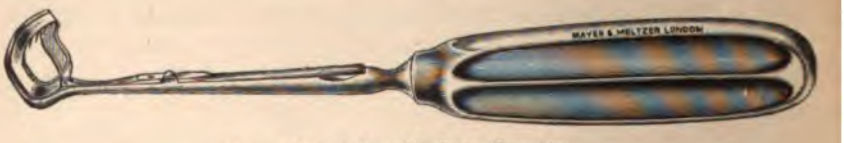

FIG. 132—Adenoid Cage Curette.

to palpate. The practised operator, however, is able to tell on curetting whether or not any fragments remain, and he avoids digital examination after the operation, in order to lessen the risk of septic infection.

2. **Removal by the Cage Curette.**—St. Clair Thomson's Modification of Delstanche's Curette is recommended. (See Fig. 132.) This is the instrument in general use.

With the patient recumbent, the instrument is passed over the tongue and behind the soft palate, care being taken not to injure the uvula, for which reason some insert it turned on the side. It is then carried up to the roof of the naso-pharynx, and, being held as a dagger is grasped, with the end emerging from the ulnar side of the closed hand, it is pressed back until it is felt to be in firm contact with the posterior pharyngeal wall, swept down in this contact, and then brought sharply out over the tongue. The whole manipulation is effected by one single turn of the wrist, and elevation of the elbow, which brings the curette through a semicircle, the curve of which is approximately that of the posterior pharyngeal wall.

With the patient seated.—The instrument is held in the palm of the hand like a dinner-knife, and the sweep down is effected by a downward movement of the forefinger coupled with a rise of both wrist and elbow.

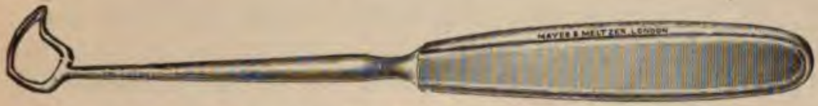

FIG. 133.—Simple Adenoid Curette.

The adenoids are impaled upon the hooks of the instrument, and grasped also by the spring-held cage.

The operation is completed by free rapid strokes of the simple adenoid curette (Fig 133), particular attention being paid to the sides of the naso-pharynx.

Tags.—If the cage-curette is allowed to become blunt, the adenoid mass is detached above, but escapes as the curette reaches the lower part of the naso-pharynx, and hangs down in the pharynx until removed by forceps. Short of this, smaller pieces of adenoid tissue or of mucous membrane may be stripped from the posterior wall, and escaping notice at the operation, may remain as semi-detached " tags " until accidentally discovered at some later date.

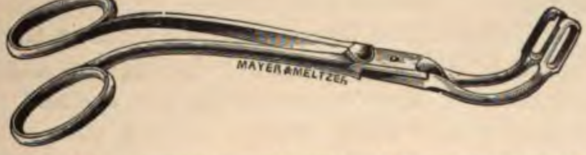

FIG. 134.—St. Clair Thomson's modification of Jurasz's Adenoid Forceps.

They may be quite safely left for two or three weeks, if not very large, as they usually slough off or ulcerate away.

If they have to be removed, seize them with Jurasz's forceps (see Fig. 134) and twist them off by a movement like turning a key in a lock. If torn off, the mucosa may strip and come away in a tongue.

3. The whole operation may, indeed, be performed by *Jurasz's forceps* (Fig. 134), which are quite safe for the purpose, except in infancy, when the posterior edge of the septum is still oblique, and when the forceps may grasp the septum, and remove part of it.

Choice of Instrument.—In the writer's opinion the La Force Adenotome is unquestionably the best instrument, and for the following reasons:—

1. The adenoids are removed from below upwards without stripping the mucous membrane.
2. Tags are never produced.
3. There are no sharp hooks to tear the uvula or soft palate.

It is most useful in children and when the adenoids are soft. But in adults with tough fibrous adenoids smooth on the surface and firmly attached, the sliding knife is apt, unless hard pressure be maintained, to glide over the surface and to leave portions of the mass behind.

In curetting, excess of zeal is to be deprecated. There is no need to expose the pre-vertebral fascia, still less the bodies of the vertebræ.

Pay particular attention to the sides of the naso-pharynx, in order to clear the Eustachian orifices of the growths.

Hæmorrhage at the moment of operation is sharp, but it ceases on sluicing the face with ice-cold water.

The total quantity of blood lost by a child in the complete tonsil-adenoid operation seldom exceeds three or four ounces.

Continuation of hæmorrhage from the adenoid wound is generally due to incomplete removal of the adenoids. The first step in checking it, therefore, is to curette the naso-pharynx once more. If this fails, a marine sponge, attached to a string, and inserted into the naso-pharynx, will almost invariably control the flow. It is worth noting that the cavity of the naso-pharynx can accommodate quite a large sponge even in children.

I have only once found it necessary to pack the naso-pharynx with gauze rolled up into an oval pad, and held in position by a string through the nose.

If the hæmorrhage does not appear for some hours or days after the operation, the method of procedure is the same, but the reader is referred for further details to the section on tonsil hæmorrhage at p. 60.

If in haste for a diagnosis of the source of the blood, make the patient hold the head forward with mouth open. In this position, blood from the adenoid wound emerges at the nose; blood from a tonsil wound at the mouth.

Difficulties and Dangers.—The chief difficulty consists in effecting the removal of the whole of the adenoids, those at the most superior recess of the roof, and those in and about the fossæ of Rosenmüller being the most liable to escape.

Practically the only danger is septic infection of the wounds, and this can be eliminated by the careful pre-operative toilet of the mouth. (See p. 52.)

Sepsis of the wound readily leads to infection of the middle ear, and acute suppuration with all its attendant complications and sequelæ may follow the adenoid operation. It is, indeed, the most frequent complication of the operation.

Otherwise, septic infection of the adenoid wound has been known to lead to septic meningitis and death, but such catastrophes are extremely rare.

The *After-Treatment* consists in keeping the patient in bed for three days, and indoors for a week. Unless the tonsils have also been removed, no gargling, douching, or other local interference is called for. The patient should not blow the nose, save one side at a time, for a week after operation.

Results.—We have already discussed above the results of the operation, as well as the chances of recurrence.

It is necessary also to add that cases are occasionally encountered in which, in spite of successful operation, the child still continues to use the mouth in breathing, and still continues to snore. As a rule, the remedy for this bad habit consists in well-devised breathing exercises carried out regularly. But sometimes the habit persists in spite of all our efforts to overcome it. For such cases, the plan of wearing an obturator across the mouth at night may be tried. Occasionally, however, it will be found to be due to the adenoids having been incompletely removed!

The fond belief that adenoids may be induced to vanish away by practising respiratory gymnastics should be denounced. It merely leads to a waste of valuable time.

Naso-pharyngeal Catarrh (**Post-nasal Catarrh**) generally occurs in association with pharyngitis or with nasal catarrh or suppuration, especially of the sphenoidal sinus. When in the course of an acute or chronic nasal or pharyngeal catarrh the naso-pharyngeal symptoms predominate, the cause will generally be found in the presence of adenoids, more or less atrophied. Post-nasal catarrh is essentially a disease of adult life, yet it is, from one point of view, none the less true that children, the subjects of adenoids, are constantly being attacked by pharyngeal, nasal, and post-nasal catarrh.

Thus, post-nasal catarrh is in reality a secondary effect of some other disease, and the *treatment* necessary is the removal of the cause.

Tuberculosis and Lupus limited to the naso-pharynx are extremely rare, but tuberculomata attached to the roof or posterior wall, and removed under the impression of their being adenoids, have been recorded.

Syphilis.—Gummata manifest a preference for the posterior or postero-superior surface of the soft palate, and also for the posterior wall of the naso-pharynx. They cause signs of "post-nasal catarrh," sometimes with obstructive symptoms, such as snoring.

On examination of the pharynx, the redness and swelling of the soft palate should lead to an examination of the naso-pharynx by posterior rhinoscopy, or with the endoscope, and to the discovery of the lesion.

It is to be distinguished from an epitheliomatous ulcer by its sharp non-infiltrated edge, its wash-leather base, and its prompt response to treatment.

The *treatment* is detailed at p. 71.

Obliteration of the naso-pharynx from *syphilitic scarring* is discussed at p. 24.

NEOPLASMS OF THE NASO-PHARYNX

(Naso-pharyngeal Fibroma)

Fibroma is the name given to the most common simple tumour of the naso-pharynx. Springing from the basi-occiput or basi-sphenoid, it is composed of fusiform cells and immature fibrous tissue interpenetrated with wide, thin-walled blood-vessels, in virtue of which it is extraordinarily vascular.

In its growth, the tumour fills the naso-pharynx, and grows into the nose, and may, if allowed to develop, reach to the orbits, the pterygo-maxillary fossæ, the antra, and the cranium, producing great facial deformity, and finally causing death.

Fibromata are commoner in males than females, and appear as a rule in early puberty. After several years of activity, their growth often comes to a standstill about the age of twenty-one, and they may even undergo involution if the patient survives the period of their growth.

We have already indicated that the naso-pharyngeal " fibroma " and the " sarcoma " of the ethmoidal region have many features in common, and that they may, perhaps, be regarded as akin to each other. (See p. 294.)

Symptoms.—The earliest symptoms of respiratory obstruction and hæmorrhage generally lead to their discovery and removal before they reach a size large enough to cause facial deformity.

A fibroma is liable to be mistaken for adenoids, but it is much firmer to the touch, and is smooth on the surface.

Treatment.—Operative Removal is necessary, and the ordinary operation is often one of considerable difficulty and danger from the very free hæmorrhage.

1. When the tumour is small in size, it is frequently possible to remove it by passing through the nose a long, strong periosteal elevator. This, guided by the finger in the naso-pharynx, is insinuated under the periosteum of the basi-sphenoid or basi-occiput, and the tumour is raised off the bone along with the periosteum. If this can be successfully accomplished, removal will be effected with a minimum of bleeding.

2. If we have to deal with a larger tumour, a more roomy access may be necessary. For this, Moure's lateral rhinotomy (see p. 295) generally suffices. The same principle in separating the tumour, should, in all cases, be followed; namely, to work, if possible, under the periosteum, and to effect the removal with as much speed as can be attained. As in the ethmoidal tumours (see p. 298), bleeding ceases as soon as the tumour tissue has been entirely removed.

The free hæmorrhage during operation renders a preliminary laryngotomy advisable. The anæsthetic is given through the laryngotomy tube, and the laryngo-pharynx is packed with sponges or gauze, to prevent the ingress of blood into the larynx and trachea. For these cases, indeed, ether anæsthesia by the intratracheal method has proved to be very serviceable.

If the patient has been reduced by previous hæmorrhage, and if the tumour has grown extensively, and made connections elsewhere, the operation may be one of great danger.

Ligature of the external carotid is sometimes performed, as a preliminary step.

If hæmorrhage continues or recurs after the operation, the tumour has been incompletely removed.

3. *Diathermy.*—Here, also, as in pharyngeal cancer, diathermy bids fair to replace the older methods of operating, as the growth can thereby be removed or destroyed with little or no bleeding.

Results.—Thoroughly extirpated, naso-pharyngeal fibroma does not recur.

CANCER OF THE NASO-PHARYNX

Epithelioma, sarcoma, and malignant endotheliomata occur in the naso-pharynx.

Symptoms.—A common early symptom is the onset of deafness or of suppuration in one middle ear. Otorrhœa appearing for the first time in a male over middle age should always raise the question of naso-pharyngeal cancer.

Another symptom, noted before the suspicion of tumour may arise, and due to the implication of nerve-trunks, is the occurrence of " neuralgia " in the course of the first or second division of the fifth cranial nerve, with paræsthesia and anæsthesia of the areas it supplies.

Finally, the local symptoms of respiratory obstruction, fœtid discharge, and infiltration of the palate set in.

If the growth begins on the upper surface of the soft palate, the foregoing nerve symptoms are absent, and the patient's only complaint will arise from nasal obstruction and deafness.

The diagnosis is made on the discovery of the tumour mass in the naso-pharynx by endoscopy, posterior rhinoscopy, or palpation.

Treatment.—When the growth is situated on the posterior or lateral wall it can only be partially removed, and if operated on, will recur locally.

When it is confined, however, to the soft palate, the growth may be entirely removed by

REMOVAL OF THE SOFT PALATE (STAPHYLECTOMY)

Indications.—Malignant growths limited to the soft palate, especially on its upper and posterior surface.

Preparation is the same as for other throat operations, the mouth being cleansed with care.

Anæsthetic.—Chloroform, administered on a mask, and, after the operation is begun, by means of the Junker apparatus.

If the operation is effected by cutting instruments, a preliminary laryngotomy should be performed, and the anæsthetic administered through the laryngotomy tube, the introitus laryngis being guarded by captive sponges.

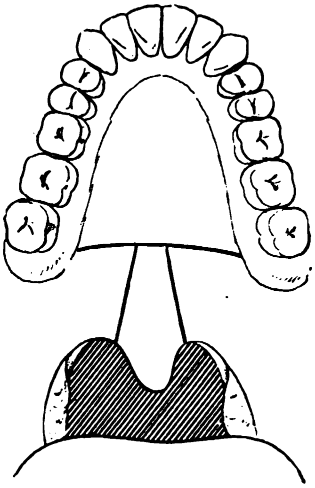

FIG. 135.—Operation for Removal of Soft Palate. Incisions.

Operation.—The mouth having been gagged open, a stitch is passed through the tongue, and given to an assistant to hold.

The incisions made are three in number (see Fig. 135), one transverse across the roof of the mouth, a little posterior to the posterior margin of the bony palate, and one on either side of the uvula, running from the arch of the velum to join the transverse incision. These incisions are deepened and carried right through the palate so as to isolate and separate the uvula and the median segment of the soft palate.

If the incisions are made with the diathermy terminal, or failing that, with the dull red galvano-cautery, there will be little or no bleeding. Otherwise, the hæmorrhage will be fairly smart, but it can be controlled by pressure-forceps and sponge-pressure.

The use of a diathermy or cautery blade permits also of the incisions traversing the substance of the growth since the cells lining the cautery wound are destroyed by heat, and are thereby sterilized. If a cold cutting blade be used, however, the incision must skirt the growth, or living cancer-cells may be transferred by the knife to healthy tissue.

The medial segment of the palate having been removed, the lateral portions remain. For hæmostatic reasons, they should be removed with the Lermoyez tonsil snare, the wire-loop being tightened and closed very gradually. (Fig. 13.)

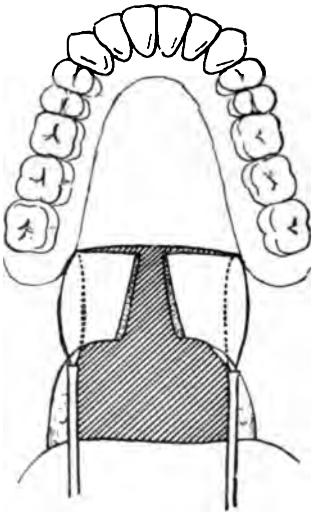

FIG. 136.—Operation for Removal of Soft Palate. The mesial segment including the uvula has been removed to permit of application of the Lermoyez tonsil snare to the lateral portions.

Diathermy and the galvano-cautery are unsuited for the removal of the lateral pieces, as secondary hæmorrhage is prone to occur during the separation of the cautery sloughs from the side of the palate where the vessels enter. Such secondary hæmorrhage is less liable to occur if the cold snare is used, and if the wound surfaces can be kept free from sepsis during convalescence.

In cancer of the soft palate, the tonsils should be removed at the operation, as recurrence may make its appearance in these glands.

After-Treatment.—During the first few days, swallowing is difficult, as liquids tend to come back through the nose. This the patient can prevent by holding the nose during deglutition. In any case, the power of swallowing is soon regained.

Speech, however, remains nasal during the rest of the life, although it may be improved by a suitable obturator.

The *Results* depend upon the success with which the tumour can be removed.

Partial Staphylectomy is permissible when the growth is situated on the buccal surface of the soft palate, and is of small extent. The opposite cheek may be split to facilitate access. The growth should be removed in one piece surrounded by a ring of healthy, non-infiltrated tissue.

CHAPTER XI

EXAMINATION OF THE EAR

FUNCTIONAL EXAMINATION

In the ear there are two organs of special sense, that of hearing located in the cochlea, and that of equilibration situated in the semicircular canals—the vestibular or canalicular system. (Figs. 137, 142.) Each of these systems possesses its own proper end-organ, nerves, and nerve-centres. Consequently, the functional examination of the ear comprises the testing both of the cochlear system and of the vestibular system.

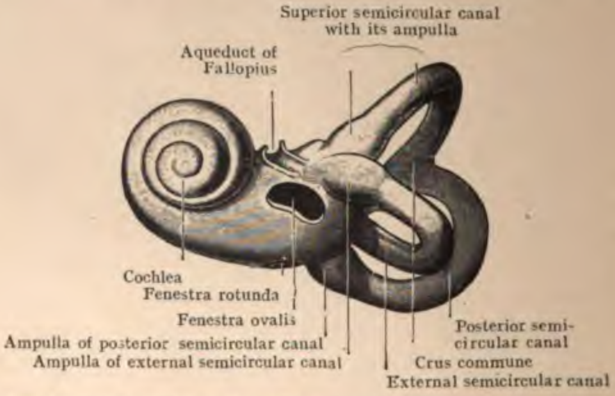
Superior semicircular canal with its ampulla
Aqueduct of Fallopius
Cochlea
Fenestra rotunda
Fenestra ovalis
Ampulla of posterior semicircular canal
Ampulla of external semicircular canal
Posterior semicircular canal
Crus commune
External semicircular canal

FIG. 137.—Left Bony Labyrinth viewed from outer side (Cunningham's Textbook of Anatomy).

COCHLEAR SYSTEM.—HEARING

The auditory apparatus consists of two mechanisms: First, that for the reception and transmission of sound-waves to the end-organ—*the conducting apparatus*; secondly, the end-organ of hearing itself in the cochlear section of the labyrinth, which, together with the cochlear nerve, tracts and centres in the brain, constitute *the perceptive apparatus*.

The tests employed in the examination of hearing have a double object. First of all, they seek to estimate the amount of hearing present, or, to put the matter in another way, they seek to estimate the severity of the deafness in a deaf patient. Secondly, they inform us whether it is the conducting apparatus or the perceptive apparatus that is chiefly or solely at fault.

Deafness due to interference with the conducting apparatus—*obstructive deafness*—presents, as we shall see later on, certain characteristics which enable us to distinguish it from deafness due to interference with the perceptive apparatus—*perceptive deafness*, or *nerve deafness*.

That is to say, we are able to determine, from the findings obtained on testing the hearing of a deaf person, whether the lesion producing the defect is situated in the conducting apparatus or in the perceptive apparatus, or in both.

The conducting apparatus, interference with which causes obstructive deafness, comprises the external auditory meatus, the tympanic membrane, and the chain of ossicles (malleus, incus and stapes), the movements of which are incommoded by lesions, not only in the tympanic cavity, but also in the Eustachian tube. When the hearing-tests manifest the characteristics of obstructive deafness, therefore, we know therefrom that the lesion must lie somewhere in those structures.

In like manner, when the hearing tests show the deafness to be perceptive deafness (or nerve deafness), we know that the lesion must be one affecting the cochlea, or the cochlear branch of the auditory nerve, or its tracts or centres in the brain.

HEARING TESTS

Conversational Voice.—As the penetrating quality of the voice varies considerably in different individuals, the results obtained when different observers test the same patient must also vary. But with the same observer, fallacy will be minimized in so far as a comparison of the hearing of a patient on repeated occasions is concerned, if the examiner employ tones and words as nearly uniform as possible in pitch and loudness.

Method.—The patient stands half-sideways to the examiner with the ear to be tested towards him, the other ear being stopped with the finger. The examiner withdraws to a distance of about twenty-four feet, and utters the test-word or sentence, the patient being asked to repeat what he hears. If the word is not correctly repeated at twenty-four feet, the examiner takes a step nearer, and again utters the word; and so on until the point is reached at which the patient is able to repeat correctly what has been said. The distance intervening between examiner and patient is then noted, in feet or inches, as the case may be.

Some words are more easily heard than others; those with many sibilants, for example, carry a long distance, and should, therefore, be avoided in making the tests.

Words like "America," "potato," "Calcutta," and numbers like "four-hundred-and-fifty-two," and so on, are the most suitable.

If words uttered in a conversational voice can be correctly heard at about twenty or thirty feet, the hearing for the voice may be regarded as normal. Any distance less than that indicates defect, as a rule. In noting down the result of this test, the examiner, of course, should only record the number of feet (or inches) at which the patient hears the test words.

Whisper.—The whisper is more uniform than speech, so the results obtained by using the whispered word instead of the spoken word are more reliable. In examining any given case, however, both should be employed. The normal hearing distance of a whisper in a quiet room is about twenty feet; that of spoken words is greater, of course, but much more variable.

We employ the "gentle" whisper, and not the "forced" or "stage" whisper.

Method.—The same as that for the voice, the only difference being that the test-word or sentence is whispered, instead of being spoken aloud.

Acoumeter or Watch.—The Acoumeter is an instrument constructed to produce a clicking or tapping sound. Scientifically, it is superior to a watch, as all acoumeters of the same pattern produce a sound of the same character, whereas, of course, watches vary enormously in the loudness of their tick.

In spite of that fact, if the furthest distance be known at which the tick of any particular watch is audible to the normal ear, the watch-test will provide a fairly accurate estimate of the hearing power for clicking sounds.

Method.—Hold the watch beyond the furthest point at which it is normally audible, and approximate it gradually to the patient's ear. The patient's eyes should be closed. Note the distance (in inches) of the watch from the ear when the patient first detects the tick. The finding should be written as a fraction: e.g., "$\frac{6}{32}$" means that a watch normally audible at thirty-two ins. is not heard by the patient when beyond six ins. from the ear.

Tuning Fork Tests.—In the ordinary way we now pass on to the tuning-fork tests. The fork generally used for *quantitative* tests is that corresponding to middle C on the piano (making 256 V. per second). (See Fig. 138.) The standard of comparison is the observer's own hearing power, provided that that is normal or nearly so. If abnormal, allowance must be made for the degree of abnormality in registering the results of the tests.

The tuning-fork used in the ordinary tests is provided with a flattened, disc-shaped end, to enable it to be closely applied to the mastoid region.

Low-toned forks are also employed, as we shall see later.

The tuning-fork is used to test the patient's hearing for sounds transmitted, first, through the air, the external meatus, the membrane and ossicles; secondly, through the bones of the head, as it is by a comparison of those two routes of sound-conduction that a distinction can be drawn between obstructive deafness and nerve deafness.

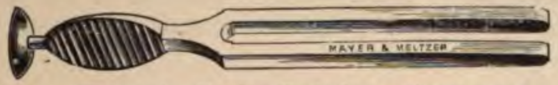

FIG. 138.—Tuning Fork for testing hearing.

Thus, the tuning-fork tests are qualitative as well as quantitative.

Tests for Hearing by Air-Conduction.—*Method.*—Sound the fork by striking one of the prongs, near its proximal end, against your knee. Do not strike the fork against a table or other hard substance, and avoid striking the free end of the fork, for in so doing, overtones are generated by the irregular or uneven vibration. While it is sounding fairly loud, hold it close to the auditory meatus of the ear you are testing in such a way that one prong of the fork is in line with the other and with the meatus. Do not hold the fork obliquely towards the ear, or the vibrations of one prong may neutralize those of the other, and the patient will hear nothing. Make the patient tell you the moment he ceases to hear the sound, then rapidly transfer the fork to your own meatus, and, with your eye on the watch, observe the difference in seconds between your own hearing and that of the patient. If the patient does not hear the sound as long as you do, note the difference in seconds, preceded by the *minus* sign, thus: Meatus = −12″, or −15″, as the case may be. If the patient hears it as long as you do, and your hearing is normal, indicate the result by the mathematical sign, ±. If he hears it longer than you do, precede the number of seconds with a *plus*, so: Meatus = +5″, or +10″, or whatever it may be. In this way the hearing for the aerial conduction of sound is tested.

Test for Hearing by Bone Conduction.

This and the following tests are primarily *qualitative*, inasmuch as their chief use is to differentiate obstructive from nerve deafness. This distinction depends upon the fact that *in obstructive deafness, the bone-conduction of sound is greater than normal.* In nerve deafness it is less than normal.

Method.—Bring the disc-shaped end of the vibrating tuning-fork into close contact with the mastoid of the ear being examined. When the patient ceases to hear it, transfer it to your own mastoid, and note whether or not you hear it. If you do, then the patient's bone-conduction is diminished. Note, as in testing the air conduction, the difference in seconds, and express the finding as mastoid = −8″, or whatever it is.

Of course, both in this test and in the last, if the patient's hearing is more acute than your own, hold the fork, to begin with, at your own ear until the sound is no longer audible by you, and then carry it to the patient's ear.

If, then, you cannot hear the sound when the patient ceases to hear it, strike the fork again, hold it on your own mastoid, and when the sound stops, apply it to the patient's mastoid. If he is now able to hear it, his bone-conduction is increased; the finding is written in seconds with a *plus* sign, thus: mastoid = +8″.

We have already seen that a decreased bone-conduction (*minus* mastoid) denotes nerve-deafness, while an increased bone-conduction (*plus* mastoid) denotes obstructive deafness.

The next two tests (Rinne's and Weber's) do not introduce any new principle into the examination, but they are useful in emphasizing and rendering clearer the difference between the air-conduction and the bone-conduction.

Rinne's Test is the more important of the two. By it we compare the air-conduction with the bone-conduction of the same ear.

Method.—Apply the vibrating fork to the patient's mastoid, and when he ceases to hear it, transfer it to the meatus of the same ear without again striking it, and ask him if he can hear it there. If he can, note how many seconds elapse before he ceases to hear it, express the difference between the two in seconds, thus: + n", or —n", as the case may be. If the air-conduction is more prolonged than the bone-conduction, Rinne's test will be + so many seconds. If the air-conduction is less than the bone-conduction, Rinne's test will be – so many seconds. In the latter case, of course, we begin the test by holding the fork opposite the meatus, and when the sound is no longer heard, we bring it into contact with the mastoid. In doubtful cases, which are not uncommon, it is advisable, while the fork is still vibrating fairly loud, to keep on applying it, first to meatus, then to mastoid, alternately, until the patient says he no longer hears it in one or other position.

It should be noted that when the hearing is *normal*, Rinne's test is *positive*.

In *obstructive deafness* (testing with tuning fork 256 V.) Rinne's test is *negative*.

In *nerve-deafness*, Rinne's test is *positive*. *

The Vertex or Weber's Test compares the *bone-conduction of the one ear with the bone-conduction of the other ear*.

Method.—Sound the fork; place the disc in firm contact with the vertex of the patient's head, and ask him in which, if either ear, he hears it the louder or the longer. If the hearing power in the two ears is decidedly unequal, the sound will be referred to the deafer ear in obstructive deafness (because the bone-conduction will be greater in that ear); while in nerve deafness, on the other hand, the sound will be lateralized to the better ear.

This test is often unreliable, as it depends upon the ability of the patient to analyse his own sensations accurately. Many patients with undoubted obstructive deafness in one ear, declare they hear the fork better in the sound ear because they expect to do so.

In carrying out these tests in cases where one ear is much more deaf than the other, it is proper when examining the aerial conduction to stop the ear not being tested with the finger; but in the tests concerned with bone conduction, this must not be done, or the results will be vitiated.

Summarizing the foregoing, we may say that by testing the air-conduction (tuning-fork at the meatus) we investigate the condition both of the conducting and of the perceptive mechanisms. By testing the bone-conduction (disc of tuning-fork in contact with mastoid region) we interrogate the perceptive mechanism chiefly. By Rinne's test (tuning-fork alternately at the meatus and on the mastoid), we compare the conducting apparatus with the perceptive apparatus of the same ear. And by Weber's test (tuning-fork on the top of the head), we compare one ear with the other.

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* Estimated by means of the monochord Rinne's test is said to be always negative, even in nerve deafness.

In the tests about to be described, the *Qualitative Tests*, we endeavour to ascertain the upper and lower limits of the patient's hearing power for musical tones.

Estimation of the Upper and Lower Limits of Hearing.—In health, the upper tone limit reaches as high as 40,000 V. per second, in childhood and adult life. After sixty, however, it is lowered, a fact which must be allowed for, in testing. In nerve-deafness there is a pathological lowering of the upper tone-limit, while the low tones are still audible. In obstructive deafness, on the other hand, the deep tones are always lost, while the shrill tones are frequently, though not invariably, audible.

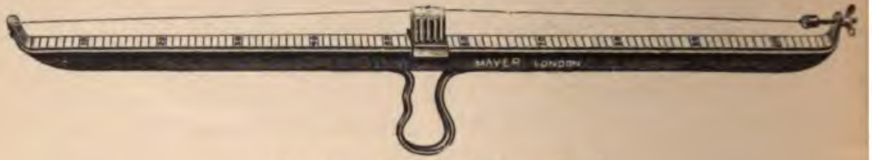

FIG. 139.—Schultze's Monochord.

Estimation of the Upper Tone Limit.—For this purpose, Schultze's *monochord* is the most accurate—indeed the only accurate test-instrument. It consists of a metal wire, which emits very high tones, the shriller tones being evoked by shortening the length of the vibrating cord, as if it were a stringed musical instrument. (Fig. 139.)

The instrument in common use, however, for the estimation of the hearing for the upper tones is the *Galton Whistle* (Fig. 140) as modified by *Edelmann*, and for ordinary purposes it is reliable enough to aid materially in differentiating between obstructive and perceptive deafness.

This instrument produces a shrill tone, which can be raised or lowered in pitch as desired. Markings upon the stem of the instrument correspond approximately to the number of vibrations produced at the different levels.

Method.—Screw the obturator that reduces the length of the tube down to the end where a whistle of the lowest note is produced. Make the patient block up with his finger the ear not being tested. Warn the patient not to listen to the blowing sound emitted by the instrument, but to pay attention to the whistle only. When the lowest whistle is sounded, ask him if he can hear it. If he can, keep on sounding the whistle while you screw the obturator upwards, so as to render the sound more and more shrill, and tell the patient to inform you when he no longer hears it. Then reverse the movement of the barrel, and ask the patient to intimate when the shrill sound returns. This may have to be repeated several times to obtain a satisfactory answer.

Then inspect the graduated marks on the stem of the tube, marking the highest spot at which the patient can hear the whistle, and note the finding, so :—" Galton=3.4, or 2.4," as it may be.

Over the age of 50 years a Galton of 1 or 1.5 need not be regarded as pathological. But in earlier years such a reading, and *à fortiori*, a larger figure is to be looked upon as abnormal.

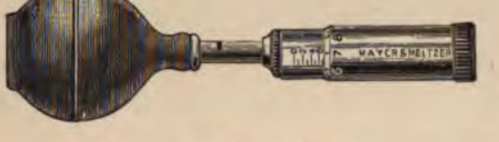

FIG. 140.—Galton's Whistle.

A decided lowering of the upper tone limit, as estimated by the Edelmann-Galton Whistle, in combination with a diminished bone-conduction for the tuning-fork, indicates nerve-deafness.

As a scientific instrument, the whistle is unreliable. But for the rough testing of clinical examination, it is probably good enough.

Estimation of Low Tones.—Loss of hearing for the deep tones is caused by obstructive deafness. For this part of the examination the deep tuning-forks (from 64 to 128 V.) are employed. In testing, the air-conduction only is interrogated.

SYNOPSIS OF FINDINGS

Summing up the results of the foregoing tests, we diagnose, first, the amount of deafness present by (1) the furthest distance at which the patient can hear the voice, the whisper, and the watch-tick; and (2) by the amount of reduction for tuning-forks of a middle tone as compared with the normal. Secondly, with regard to the type of deafness present, we consider the deafness to be **obstructive** (the lesion in the conducting apparatus) if :—

(1) The bone-conduction exceed the normal (*plus* mastoid) ;

(2) The bone-conduction is increased relative to the air-conduction (*minus* Rinne) ;

(3) The hearing for low tones is abolished or reduced ;

(4) With the tuning-fork on the vertex of the head, the sound is lateralized to the deafer ear.

On the other hand, we consider the deafness to be **perceptive or nerve-deafness** (the lesion in the nervous apparatus) if :—

(1) The bone-conduction is less than the normal (*minus* mastoid) ;

(2) The bone-conduction is decreased relative to the air-conduction (*plus* Rinne) ;

(3) The upper tone limit is lowered, while the lower tones are audible ;

(4) With the tuning-fork on the vertex, the sound is lateralized to the better ear.

Note that the difference between these two varieties of deafness rests upon (1) the results obtained by comparing the patient's bone-conduction with the normal bone-conduction ; (2) the results obtained by comparing the patient's bone-conduction with his air-conduction ; and (3) the results obtained by testing with Galton's whistle (or Schultze's monochord) and the deep forks. In obstructive deafness, the bone-conduction is increased as compared with the normal and with the air-conduction ; in nerve-deafness, the bone-conduction is decreased in comparison with the normal, and not increased in comparison with the air-conduction. In obstructive deafness, the upper tone limit is not lowered, while the hearing for lower tones is impaired ; in nerve deafness the upper tone limit is lowered, while the hearing for lower tones is not impaired (in mild cases). Using the technical terms, we express the difference thus : In obstructive deafness, there are present : – meatus ; + mastoid ; – Rinne ; Galton normal ; and with tuning-fork C. 128, – meatus ; in nerve deafness there are present – meatus ; - mastoid ; + Rinne ; and Galton lowered.

The following scheme is that adopted by the Otological ction of the Royal Society of Medicine for hearing tests :—

R.E. Conversational Voice *L.E.*

Single words or sentences with opposite ear closed.

Distance from ear expressed as a fraction with distance at which voice is heard from normal ear as denominator.

Whisper

Conditions as above, but whisper produced by residual air after forced expiration.

It is recognised that the voice tests are approximate only, as it is impossible to devise an exact standard. The words or phrases used to be recorded when possible.

Watch or Politzer's Acoumeter

Distance from ear expressed as a fraction with distance at which watch is heard by a normal ear as denominator. The acoumeter should be held strictly vertically.

Tuning-fork Tests

Lateralising Test (*Weber's*)

Base of vibrating fork to be firmly pressed on mid-line of forehead, and patient asked on which side it is more distinctly heard.

Relative Bone and Air-conduction (*Rinne*)

The vibrating fork is alternately pressed firmly on the mastoid and held with limbs vertical and vibrating in coronal plane close to the meatus, the position in which the tone is heard longer being noted.

Absolute Bone-conduction (*Schwabach and Bing's Tests*)

In these tests the perception of a tuning-fork pressed on the mastoid of a patient and of a normal observer is compared—(1) with the meatus open (Schwabach), or (2) with the meatus closed lightly by the finger (Bing). The difference of the duration of perception expressed in seconds between the ears of observer and patient is noted.

Tone Range

(1) (*a*) Upper limit of air-conduction by Edelmann-Galton whistle or steel monochord (gauge of wire to be stated).

(*b*) Upper limit of bone-conduction by steel monochord.

(2) Lower limit of air- and bone-conduction by tuning forks.

In all tuning-fork tests the number of vibrations per second, and whether single or double, and the make of fork to be stated in each case."

The following are examples of the case sheets in use at the Central London Throat and Ear Hospital :—

EXAMPLE I

		Right Ear.		Left Ear.
Conversation	=	1 foot	..	20 feet
Whisper	=	2 feet	..	20 feet
Watch	=	10"	..	48"
Galton	=	0.4	..	0.4
Tuning-fork (C. 256) :				
Meatus	=	−10"	..	±
Mastoid	=	+ 3"	..	±
Vertex	=		>	
Rinne	=	—	..	+
"C. 64" meatus	=	− 30"	..	±

= Obstructive deafness in right ear; left ear normal.

EXAMPLE II

		Right Ear.		Left Ear.
Conversation	=	6 feet	..	As above
Whisper	=	2 feet	..	
Watch	=	Contact	..	
Galton	=	3.4	..	
Tuning-fork (C. 256) :				
Meatus	=	−20"	..	
Mastoid	=	−10"	..	
Vertex	=		>	
Rinne	=	+	..	
"C. 64"	=	±	..	

= Nerve deafness in right ear; left ear normal.

Mixed Obstructive and Nerve Deafness.—It not infrequently happens in cases of severe deafness that the bone-conduction is reduced as compared with the normal, and yet at the same time Rinne's test is negative. In other words, the bone-conduction test indicates nerve deafness; while Rinne's test indicates obstructive deafness. The explanation is that in such cases both nerve deafness and obstructive deafness are present, the lesion affecting both the conducting and the perceptive apparatus.

Gellé's Test, when first introduced, was expected to prove of value in differentiating obstructive deafness from nerve deafness, but opinion as to its reliability is not favourable.

Method.—With the vibrating tuning-fork on the mastoid, the air in the external auditory meatus is alternately condensed and rarefied by an apparatus such as Siegle's pneumatic speculum. (See p. 428.) If the footplate of the stapes is movable in the oval windows, the increased air-pressure in the meatus, being conveyed to the labyrinth, it is said, through the movable stapes, is associated with a diminution in the intensity of the sound as heard in the ear, while decreased air-pressure in the meatus is associated with increase in its intensity. If, on the other hand, the stapes is fixed, no alteration in the loudness of the sound will be perceived by the patient.

Anomalies.—In slight obstructive deafness from mild Eustachian or middle-ear catarrh, the hearing-tests sometimes manifest some of the characters of nerve-deafness, and it is not until inflation and other methods of treatment have cured the deafness, that its real nature becomes evident. This is a warning not to rest our diagnosis, especially when the deafness is slight, solely upon the data obtained from the hearing-tests. All the circumstances of the case—the findings obtained from the physical examination of the ear and of the patient; and the effect upon the deafness produced by inflation with Politzer's bag and with the Eustachian catheter—should be taken into account before forming an opinion as to what has produced the deafness in any particular case.

Again, a warning should be uttered not to expect that the treatment of deafness, when it is partially successful, will be reflected in an improvement of the hearing for the tuning-fork. It is possible to obtain considerable improvement in the hearing for the ordinary sounds of the world without any considerable change being perceptible in the hearing for the purely musical tones of the tuning-fork. What a deaf person desires above all is to be able to hear conversation, and most otologists, therefore, reserve the tuning-fork tests for differential diagnosis alone, relying upon the voice and the whisper for the estimation of quantitative results. While that is so it should be remembered, nevertheless, that this practice, although useful and generally reliable, easily leads to fallacy, and, unless very strictly guarded, is apt to induce serious mistakes being made in the appraising of the value of methods of treatment, whether old or new.

Bárány's Noise Machine.—This instrument has a clockwork mechanism which produces a loud buzzing noise when the machine is held in contact with the ear. It is employed when absolute unilateral deafness is suspected, as in purulent labyrinthitis. The noise-machine is held against the sound ear, while a vibrating fork is applied to the mastoid of the diseased ear. If the patient is unable to hear through the noise the sound of the tuning fork in the diseased ear, it is held that the hearing in that ear is abolished: a point of importance as indicating destruction of that labyrinth. (Further information is required on this point.)

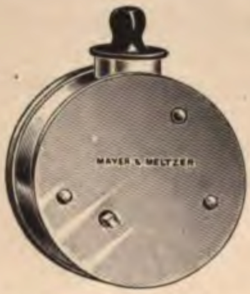

FIG. 141.—Bárány's Noise Machine.

The Noise-Machine is used in Suspected Malingering. (See p. 587.)

We pass now to consider the testing of the :

VESTIBULAR SYSTEM

(SEMICIRCULAR CANALS: CANALICULAR SYSTEM)

The activity of the vestibular system depends upon the integrity of the end-organs of the vestibular nerve in the semicircular canals which adjoin the cochlea, and also upon the integrity of the vestibular nerve-trunk, and of its centres in the medulla and brain.

We investigate the condition of the vestibular system not with the object of distinguishing between the different types of deafness, but with the idea of diagnosing the extent of disease in the labyrinth, especially in suspected purulent labyrinthitis.

The vestibular tests should be made in all cases of nerve-deafness, and also in all cases of suspected disease of the brain or spinal cord.

With regard to nerve-deafness, experience of the vestibular tests has shown that just as severe middle-ear disease, from whatever cause arising, tends to produce secondary changes in the cochlea—superimposing nerve-deafness upon the obstructive deafness—so, in like manner, cochlear lesions are often accompanied or followed by changes in the semicircular canals.

We shall preface our description of the vestibular tests by a word upon the physiology of the semicircular canals.

There are three canals in each petrous bone (see Fig. 144), and within them is situated the sensory epithelium of the vestibular system. The epithelium is ciliated, and the ciliary movement responds to the waves of the watery fluid which fills the canals, and which moves to and fro in harmony with the ever-changing position of the head and body. From this source stimuli are constantly being transmitted to the medulla, and thence by the spinal cord to the muscular organization of the trunk and limbs, and also by higher centres in communication with the nuclei of the oculo-motor nerves to the orbital muscles. Thus in health we have two sets of three canals, one set on each side supplying a constant more or less even bilateral flow of stimuli to the muscles of the eyes, and of the trunk and limbs. This even bilateral flow, among other

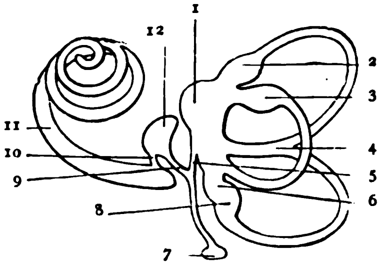

FIG. 142.—The Membranous Labyrinth. (Diagram). From Cunningham's Textbook of Anatomy.

(1) The utricle. (2) Ampulla of superior semicircular canal. (3) Ampulla of external (lateral) canal. (4) Common superior and posterior canal. (5) Ductus utriculo-saccularis. (6) Opening of posterior canal. (7) Saccus endolymphaticus (lying on the posterior aspect of the petrous, is the site of "saccus empyema"). (8) Ampulla of posterior canal. (9) Ductus endolymphaticus (passes to the saccus through the aqueductus vestibule). (10) Ductus reuniens. (11) Ductus cochlearis. (12) Saccule.

functions, maintains the equilibrium and steadiness of the body as a whole.

If, however, the stimuli are unequal; if, that is to say, the stimuli from the canals of one side preponderate in the nerve centres for any length of time over those from the canals of the other side, then a sense of loss of equilibrium—vertigo or giddiness—is produced. Along with this vertigo there appears a deviation of the muscles of the body (see later, p. 414), including those of the eyes. In the eyes the deviation is converted into a peculiar kind of nystagmus, known as *vestibular nystagmus*, and, as the vestibular tests depend upon the recognition and analysis of this nystagmus, we shall devote a paragraph to its description.

Vestibular nystagmus consists of two movements, first a slow deviation of the eyeballs in one direction, followed by a rapid twitch of the eyeballs back again to their original position. These unequal oscillations follow each other rapidly, and, once seen, are unmistakable.

A further peculiarity of vestibular nystagmus is this, that the nystagmus is exaggerated when the person turns his eyes in the direction of the rapid twitch, and it is diminished or abolished when he turns his eyes in the direction of the slow deviation. For this reason we speak of the nystagmus as being *directed* to the side of the rapid twitch. Thus, *nystagmus "to the right"* means that the *rapid twitch is directed to the right*, and that the nystagmus is increased on turning the eyes to the right, and *vice versâ*.

Vestibular nystagmus, as we have seen, together with vertigo, is produced when the canalicular stimuli from one side preponderate over those from the other side. This condition is fulfilled in certain diseases of the labyrinth. (See later, p. 417.) If, for example, a sudden pathological blow, such as hæmorrhage or acute suppuration, destroys the vestibular end-organs on one side and paralyses their function, then we find that there is *spontaneous* vestibular nystagmus to the healthy side when we examine the eyes.

But the condition may also be fulfilled, and vertigo and nystagmus produced, by excessive artificial stimulation of healthy semicircular canals. The methods by which we can artificially bring about these results are (1) rotation of the body, (2) heating or cooling the external auditory meatus by hot and cold water, and (3) galvanism.

It is the first two methods which we habitually employ in our clinical tests of the activity of the vestibular system.

THE VESTIBULAR REACTION TESTS

Rotation.—*Method.*—(See Fig. 143.) The patient is seated with his feet clear of the ground in the rotating chair or stool. His eyes should be covered with shaded spectacles. He is then rotated with the head erect in one direction at the rate of ten revolutions in twenty seconds. After ten revolutions, the rotation is suddenly stopped, and the eyes at once examined. (Fig. 144.)

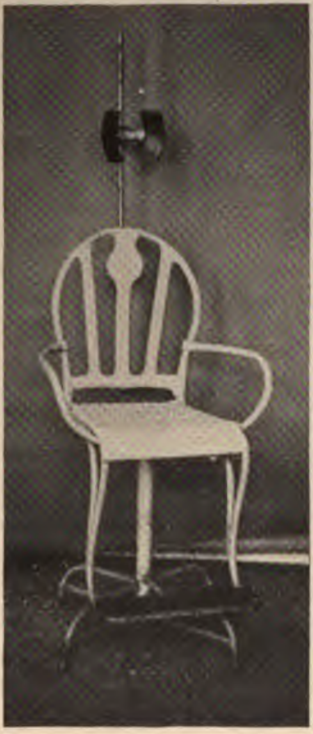

FIG. 143.—Author's Rotating Chair.

If the vestibular system is normal, there will be well-marked nystagmus affecting both eyes with its direction (i.e., the direction of the short twitch) *towards the side from which the patient was rotated.* (Fig. 144.)

Thus, if the patient is rotated from right to left, the nystagmus, after rotation, will be directed towards the right, and *vice versâ.*

Further, if the duration of the nystagmus, after rotation, be measured, it will be found to last about thirty seconds, and to subside gradually. Definite but not excessive vertigo is experienced for a few moments after the cessation of the rotation. It passes away before the nystagmus disappears.

Significance of Results.—In our analysis of these phenomena we do not propose to follow the subject into any of its intricacies. This bald statement alone will be made: namely, that while rotation necessarily stimulates the semicircular canals of both sides, one side is more affected than the other, and the side which is the more affected is the one towards which the nystagmus is directed. Thus, if the nystagmus is directed towards the right,

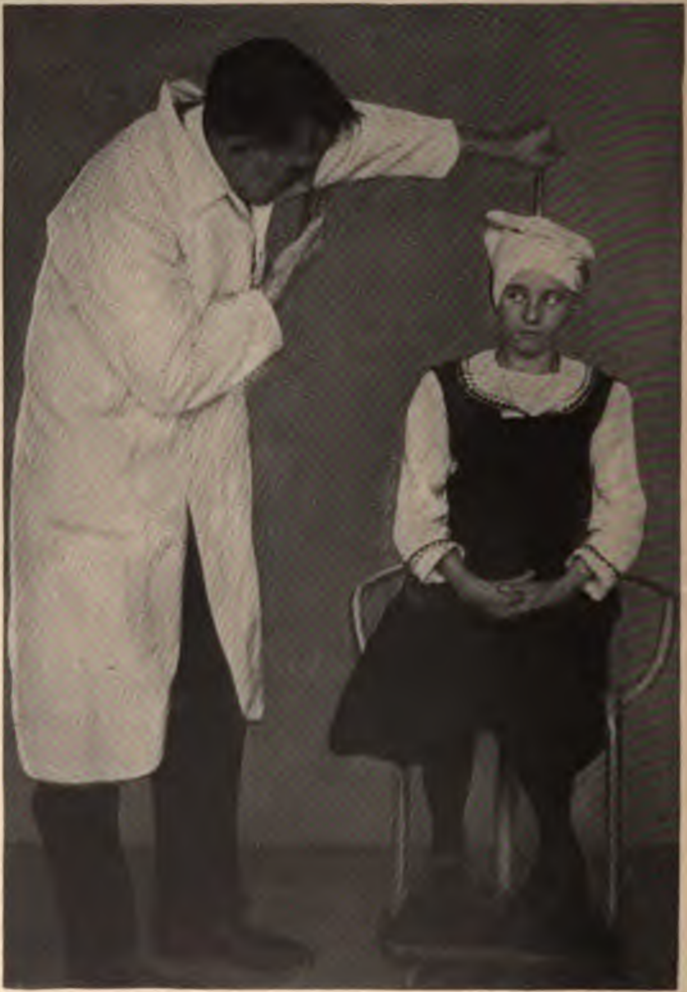

FIG. 144.—Patient in the Rotation Chair, after rotation from right to left to test the integrity of the right labyrinth. (Strictly speaking, the patient should not be encouraged to fix the eyes on any object.)

we know that it is the right labyrinth which is being chiefly stimulated.

To put this statement in a more convenient way, we may say that, as there is nystagmus to the right after rotation from right to left, when we wish to ascertain the condition of the semicircular canals on the right side, we rotate the patient from right to left. And on the other hand, when we wish to test the canals on the left side, we rotate the patient from left to right, and look for nystagmus to the left.

So much for the normal reaction. We come now to discuss the effects of rotation when, first, the vestibular system of one side is inert, and secondly, when the vestibular system on both sides is inert.

When the vestibular system of one side is inert, the other being active, rotation from the diseased side will be followed by a feeble response, while rotation from the healthy side will be followed by a normal response, for the following reason: We have seen that while rotation necessarily stimulates the canals of both sides, nevertheless it affects one side more than the other. For this reason, in unilateral vestibular destruction or paralysis, rotation to either side will be followed by nystagmus. But we have also seen that after rotating the patient from right to left (for example), it is the right labyrinth that is chiefly stimulated. If, then, this labyrinth is destroyed or paralysed, rotation from right to left will stimulate the left labyrinth only, and its stimulation will only have a feeble result—the nystagmus, as a matter of fact, lasts only about fifteen seconds, and there is little or no vertigo. On the other hand, if we rotate the patient from left to right, the left labyrinth will be chiefly stimulated, and, being healthy, it will produce a normal nystagmus, lasting thirty seconds, and accompanied by vertigo.

To put the matter in another way: If rotation in one direction produces a feeble response, while rotation in the opposite direction produces a normal response, then we know that there is unilateral impairment of the vestibular system, and that *the side affected is that towards which the feebler nystagmus is directed.* If, after rotation from left to right, the nystagmus (to the left) is normal, and after rotation from right to left the nystagmus (to the right) is feeble, then the right vestibular system is at fault. And *vice versâ.*

When the vestibular system of *both sides* is destroyed, *rotation to either side* will produce *little or no nystagmus*, and *no vertigo.*

[We can alter the type of rotation nystagmus—horizontal, vertical or oblique—by altering the position of the head. This, however, is a point of physiological interest only. In describing the above tests, we have assumed throughout that the patient's head is erect.]

The Caloric Tests.—Raising or lowering the local temperature of the external auditory meatus, induces nystagmus and vertigo in health. When cold is employed, the nystagmus is directed to the *opposite* side of the ear tested: when *heat* is employed, the nystagmus is directed to the *same* side. Cold, in the form of cold water, gently injected, or run from a small douche-can into the meatus, is what is generally employed. (See Fig. 145.) Or Dundas-Grant's cold air apparatus may be used.

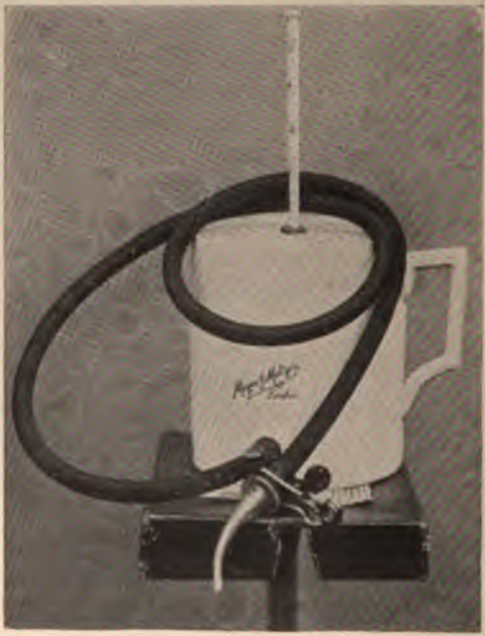

FIG. 145.—Apparatus for the Caloric Test. (Vestibulometer.)

The great advantages of this test are, that it is easily applied, and that it interrogates definitely only one ear at a time.

Method.—After making sure that the meatus is not blocked with cerumen, granulations, or polypi; water at a temperature of about 70° F.—20° to 22° C.—is slowly run into the meatus of one ear, the patient being directed to turn his eyes meanwhile to the opposite side. At the first appearance of nystagmus, the injection is stopped. In health, nystagmus will appear in about thirty seconds, and it is accompanied with mild vertigo. If the vestibular system is inert—as in suppuration of the labyrinth, etc.—little or no nystagmus appears, even after prolonged syringing. If, on the other hand, it is irritable, as in neurasthenic conditions, the reaction appears early, and is severe, being accompanied with great vertigo, nausea, and sometimes vomiting. Should such an excessive reaction set in, the unpleasant symptoms may be at once checked by the injection of warm water into the meatus.

After one ear has been tested, the other ear is likewise dealt with; but an interval of at least fifteen minutes should elapse between the two tests.

When the membrana tympani is perforated, the caloric reaction appears quickly, because the cold water comes into direct contact with the wall of the labyrinth.

We proceed now to sum up the results obtainable from the vestibular tests.

Both vestibular systems are healthy if :—

(1) Rotation to either side is followed by well-marked nystagmus directed towards the side from which the patient was rotated, accompanied by definite, but not severe vertigo, and lasting about thirty seconds.

(2) Cold water in either meatus produces well-marked nystagmus to the opposite side, accompanied by moderate vertigo (with water at a temperature of 70° F.—20° to 22° C.—the reaction appears in about thirty seconds).

(3) Hot water in either meatus induces similar results, but with the nystagmus directed to the same side.

Both vestibular systems are totally inert if :

(1) Rotation to either side is followed by no nystagmus and no vertigo.

(2) Neither cold nor hot water in either meatus induces nystagmus or vertigo.

The vestibular system of one side is inert, and the other healthy if (assuming the right side to be inert and the left to be active) :—

(1) Rotation from right to left is followed by feeble nystagmus lasting only about ten seconds, and not accompanied by vertigo.

(2) Rotation from left to right is followed by well-marked nystagmus lasting about thirty seconds, along with moderate vertigo.

(3) Cold water in the right external meatus does not induce nystagmus and vertigo.

(4) Cold water in the left external meatus does induce definite nystagmus and vertigo.

(5) The same holds good of hot water.

Partial destruction and impairment of either vestibular system may often be detected by observing the nicer details of the foregoing reactions, especially having regard to the time element, and to the type of nystagmus. When the reactions are delayed, shortened, and imperfect, although still present, a partial destruction of the vestibular system may be diagnosed.

This is especially common on old-standing labyrinth lesions affecting the cochlea and the hearing in the first place. In functional and hysterical deafness, on the other hand (in the writer's experience) the vestibular reactions are normal as a rule.

The Pointing Test.—When the balance of the vestibular system is upset by disease or by physiological stimuli such as rotation, etc., there is noticeable a deviation of the trunk and limbs away from the stimulated side. The pointing test shows whether such deviation is present or absent.

Method.—The surgeon and patient are seated face to face. The surgeon holds out his forefinger about a couple of feet above his knee. The patient places his finger upon his own knee, and then, at the word of command, raises it to touch the surgeon's finger. After doing this once or twice with open eyes, he is made to carry out the movement with closed eyes. Both sides are tested, the one after the other.

In health, the patient is able to touch the surgeon's finger without fail. But when the vestibular system is at fault, a deviation of the finger to one or other side will become evident. The rule of the deviation is as follows: Deviation takes place towards the same side as the direction of the *slow* component of the nystagmus; that is, in the opposite direction to the nystagmus. Thus, it will deviate *towards* the paralysed and *away from* the stimulated labyrinth.

(For Spontaneous Nystagmus, see p. 408.)

GENERAL SYMPTOMATOLOGY OF AURAL DISEASES

Tinnitus Aurium.—" Noises in the ear " is a very common complaint. Ascertain the character of the subjective sounds, whether " throbbing " or " singing " ; whether low-pitched, as " roaring " and " blowing," or high-pitched, as " whistling," " hissing," and so on. Ascertain also whether the noise is constant or intermittent ; whether it is aggravated or relieved by position ; whether it is associated with vertigo, or with sickness and vomiting.

Tinnitus is sometimes relieved, and sometimes it is made louder, by Politzeration.

Tinnitus is an accompaniment of nearly all affections of the conducting and perceptive mechanisms, and it may be so incessant and severe as to render life miserable. A constant high-pitched tinnitus, without any marked degree of deafness, is one of the early signs of the incurable form of deafness known as " otosclerosis."

A useful practical classification of tinnitus is into " simple " and " compound." In *simple* tinnitus, the subjective sound is uniform in character, however variable it may be in loudness. " Blowing," or " humming," or " whistling " are the usual descriptions given to this type. The milder forms accompany the milder and less obstinate forms of middle ear disease. *Compound* tinnitus consists of subjective sounds, which vary not only in loudness, but also in character, several distinct noises being simultaneously audible, often affording much mental exercise to a patient with a turn for introspection. The most usual epithets used to describe this variety are, " all sorts of noises," " whistling and puffing," " screaming." The occurrence of single loud " crashes " repeated at unexpected moments—even waking up the patient in the night—is a symptom which may produce great mental distress.

The compound tinnitus will be found in the more serious and obstinate forms of middle ear deafness. It often precedes cochlear involvement. One notes it in otosclerosis, and also in such general diseases as arterio-sclerosis and Bright's disease. Its presence should always lead to an examination of the urine.

Diagnosis.—It is important not to mistake hallucination for tinnitus aurium. In sufferers from tinnitus who are becoming insane, auditory hallucinations, no doubt initiated by tinnitus, are common. The distinction lies in this, that if the patient hears definite words, or songs, or bands playing tunes, or other combinations of sounds into which a formative or ideative element enters, then he should be regarded as suffering from an hallucination—a mental, not an auditory symptom.

Tinnitus obviously is produced by cochlear irritation, yet it is a fact that it is generally due to middle ear disease. Silent deafness is usually nerve deafness, the types of silent deafness being senile deafness, and old noise deafness. In deaf mutes and other sufferers from severe and long-standing deafness tinnitus, as a rule, is absent. The complete destruction of the cochlea by disease or by operation generally silences tinnitus, but not always.

Objective tinnitus aurium is a rare phenomenon which seems in the majority of cases to be due to a clonic spasm of palatal muscles. It bears a somewhat evil reputation as a forerunner of severe nervous or mental breakdown.

Treatment.—Subjective tinnitus aurium is, of course, only a symptom, and the best treatment naturally consists in the removal of the cause. Frequently, however, this gratifying end cannot be secured, and we are compelled to treat the symptom —it must be confessed, often with indifferent success.

Locally, the oto-masseur is often very useful, and a small hand-driven machine may be purchased by the patient himself, and attached to the domestic sewing-machine for driving purposes. The electrically driven masseur is more rapid and efficacious, but it should be retained in the otologist's hands, as, if it is incautiously used, it may do harm.

Internally.—It may be necessary to control the mental irritation produced by the tinnitus by bromides, a favourite prescription being,

℞.	Acid Hydrobrom. Dil.	♏ 30.
	Tc. Nuc. Vomic. :	♏ 8.
	Aq. ad.	℥ss.—M.

Quinine in small doses (gr.i. t.d.s.) sometimes has the effect of subduing loud tinnitus, but in moderate or large doses it is apt to cause deafness, or to make existing deafness worse.

Sometimes the vaso-dilators, such as amyl nitrite, nitroglycerine, and alcohol, relieve the tension, and render the tinnitus more tolerable. But they just as often aggravate it.

Patients with tinnitus are happiest in a noisy environment as a rule, the external uproar drowning the internal. But there are some cases in which it is rendered worse, and it may even be set up by external noise. In that event, the use of meatal plugs or stoppers is indicated.

The noises vary considerably with atmospheric pressure, humidity, and temperature. They are least troublesome in dry, warm, elevated situations, and loudest at sea-level, especially in cold, damp weather.

Sufferers from tinnitus should always be told that their symptom does not indicate a tendency to apoplexy or to insanity.

The advice sometimes given to persuade the patient to "fight against" the subjective sound has, in my experience, proved harmful, probably by the necessity for the concentration on the tinnitus that this mental manœuvre necessitates.

Finally, cases are encountered in which the tinnitus is so loud, persistent, and obstinate, that the reason may be threatened. In such cases, the mastoid and middle ear spaces should be opened and the labyrinth destroyed by the surgeon. This operation is naturally not free from danger, but the risk is not very great. A more serious criticism lies in the fact that the tinnitus may be uninfluenced by it. Nevertheless, there are a few rare cases in which it should be tried.

Paracusis Willisii is the name given to the phenomenon of a deaf person hearing better in a noisy place, like an omnibus or railway train, than in quiet surroundings. Its cause is doubtful, and so is its significance. But it is a common symptom in otosclerosis.

Vertigo or **Giddiness** is the feeling of loss of equilibrium attended with swaying, reeling, and falling, and, if severe, culminating in sickness and vomiting, which is due to irritation of the vestibular system in the ear, medulla or cerebellum. A type of ocular vertigo is also described, but does not concern us. It is to be noted that the symptom may be a mere momentary sensation of uncertainty, or it may be so severe as to render the patient prone and helpless. Consciousness is not affected, in which respect it differs from the *petit mal* of epilepsy.

Ménière's Syndrome (the labyrinth storm) is the name given to the severest types of vertigo, due to a rapidly developing lesion or functional derangement of the semicircular canals, and to a less extent of the cochlea, of one side. It is characterized by, in the order of their appearance, a low-pitched tinnitus; vertigo, at first slight, quickly becoming severe; nausea; and vomiting. The symptoms reach their height in an hour or less, and then slowly decline in the reverse order of their appearance.

Along with the vertigo, the patient is usually sensible of a subjective movement of external objects, and if the eyes are examined during the attack, they will be found to exhibit spontaneous vestibular nystagmus, usually directed to the *opposite* side from the labyrinth affected; but not invariably so.

In the decline of the attack, the vertigo and spontaneous nystagmus persist for a day or two, especially if the attack has been severe.

Any patient who has been the victim of the labyrinth storm should receive a careful examination of the ears. Not only the laity, but also medical men too often regard it as a mere " bilious attack." The same injunction applies also to the milder varieties of vertigo.

Diagnosis.—When due to disease in the ear, vertigo is usually definite: that is to say, the patient's description of the symptom is clear with a tendency to exactitude. On the other hand, vertigo from " gastric disturbances," from neurasthenia, from heart disease, from epilepsy, is less plainly observed, less clearly appreciated, and less precisely narrated.

Inasmuch as the symptom may be due to labyrinth or cerebellar disease, its occurrence in the course of middle-ear suppuration may mean serous or purulent labyrinthitis, cerebellar abscess, or meningitis.

In non-suppurative conditions, it may occur in its milder, and even in its severer forms in almost any ear disease; cerumen may induce it. Generally speaking, however, its severer forms indicate some sudden labyrinth event, such as a hæmorrhage (Ménière's historical case); a serous or purulent effusion; syphilitic disease; or herpes zoster oticus.

Severe vertigo, occasionally protracted, often characterizes shell-shock, and especially when the ears have been affected.

The severer kinds of vertigo are practically always associated with nerve deafness. And in the intervals between attacks, the vestibular tests show an impairment of the vestibular reaction. These tests cannot be applied during an acute attack.

It ought to be noted that an acute destruction of the vestibular organ in the ear produces vertigo, and so directs attention to its occurrence, but, on the other hand, slow chronic destruction may go on to finality without betraying the fact in any symptom whatever referable to the vestibular system. Such patients are, however, sufferers from severe nerve deafness. Whether the vestibular system is often destroyed independent of hearing, is at present unknown.

Prognosis.—It is remarkable that even in the milder cases in which the attack seems to be wholly vestibular there is, nevertheless, except in certain rare functional cases, a decided loss in bone-conduction, and in the hearing for high tones. This deafness persists, and in many cases, if the attacks go on, gets gradually worse until the patient's hearing is seriously impaired While this is happening, the attacks of vertigo gradually become less frequent and less severe, and finally disappear entirely. In such cases, both ears are usually affected, one more than the other, and, if tested, the vestibular reactions will be found to be impaired or absent.

Functional, or what seem to be functional cases are occasionally encountered where the attacks of vertigo and their accompanying deafness entirely disappear, and leave no trace of their presence.

And between those two extreme types, there are cases which, left to themselves, gradually get well with a certain amount of permanent deafness.

Treatment.—No treatment of vertigo should be begun until the ears have been thoroughly examined, especially with regard to suppuration, as an attack of Ménière's " disease " may be due to suppurative labyrinthitis, in which case the treatment is surgical. (See later, p. 522.) Syphilis must also be excluded (p. 580).

Severe vertigo of the Ménière type in practice, from the point of view of treatment, may be regarded as of two or perhaps three varieties, according as it occurs in patients with (1) a high blood pressure ; and (2) a low blood pressure (G. J. Jenkins) ; while in the third we may place those where it is assumed to be due to some toxæmia.

In the first variety, a restricted meat dietary, and iodide of potassium, grs. iv. to vi. t.d.s., continued for several weeks or months, will gradually lead to a disappearance of the attacks. The second variety, those with low blood pressure, are treated with ergot ; ♏ xv. to xx. of the liquid extract or *ernutin* hypodermically being used.

In the third class it is supposed that the poison is intestinal. At all events, a course of intestinal antiseptics, and especially of aperients, is often highly beneficial.

Some patients are benefited or cured by becoming vegetarian.

The Fistula Symptom.—When there is a fistula in the external bony wall of the labyrinth accessible from the external meatus, the raising of air pressure in the latter will induce vertigo. (The same may be produced, curiously enough, in children with congenital syphilitic deafness.) This test is usually applied by fitting the nozzle of a Politzer's bag accurately into the meatus, and compressing the bag firmly. If there is a fistula, the patient is at once seized with vertigo so severe that he may fall out of the chair, and with violent nystagmus. The occurrence of this sign in suppuration of the middle ear is of great value as indicating the presence of a fistula through the outer wall of the labyrinth.

Pain—Earache.—Pain in the ear is always important. It may be due to furunculosis of the meatus, and then it is severe, and is evoked or aggravated by touching and moving the auricle; to pus retention in suppuration of the middle ear; or to mastoiditis. Associated with headache, it may indicate extra-dural abscess or some other intra-cranial complication of aural suppuration. Pain in the ear without any physical sign of disease is often due to dental caries, especially of the lower wisdom or molar teeth of the same side. A rare cause of pain is cerumen impacted in the external meatus.

Discharge ("Running Ears").—Obtain the patient's opinion on the character of the discharge. Do not directly suggest names to him. Ask him what colour it is. If he says "white," ask him whether he means "clear and watery," or "thick and yellow"; if "red," whether crimson or blood-stained, or mahogany-coloured. Ascertain whether any change in the character of the discharge has been noticed—e.g., from "clear and reddish," leaving a stiff mark on the pillow, to "thick and yellow," and later, perhaps, to "reddish" again, but still "thick." This story would suggest an acute catarrhal otitis media which had become purulent from subsequent septic infection of the middle ear, and where, more recently, granulations had formed.

A discharge may be "ropy" or "stringy," from the presence of mucus in the pus.

Supposed Cause.—Although the patient's ideas on the supposed cause of his complaint may often prove to be very wide of the mark, yet in a certain number of cases information bearing on this point is of great value, and should therefore be always noted.

In tracing back the history of an ear case, note particularly—(1) the occurrence of the exanthemata, of influenza, or pneumonia, or of a family history of tubercle, in connexion with purulent discharge from the ear; (2) the occurrence of frequent colds in the head, sore throats, rheumatism and gout, in cases of chronic catarrhal deafness; (3) the engagement of the patient in any noisy employment, such as boiler-making, gun-firing, etc., in cases of nerve-deafness. Take into consideration the appearance of the patient—if it suggests anæmia, chlorosis, etc. Finally, we must not forget the possible dependence of nerve deafness upon some central nerve disease, or upon some constitutional dyscrasia, such as diabetes, myxœdema, or leucocythæmia.

PHYSICAL EXAMINATION

Having put on the forehead mirror or head-light, turn the patient's head so as to bring the affected ear opposite to you. Before inserting the speculum, examine in detail, under good illumination, the auricle, meatus, mastoid region and side of the head, the angle of the jaw, the parotid region, the sterno-mastoid, and the occipital region. Holding the upper part of the pinna between the thumb and forefinger (of the right hand in dealing with the left ear, or of the left hand in dealing with the right ear), pull it gently upwards and backwards. Direct the light on the auricle and into the meatus, and note any abnormality in the size, shape, or colour of these parts.

The Auricle.—Look for any error in development, any wound, swelling, or other deformity of the pinna. Inspect the lobule, particularly the ear-ring perforation, where occasionally lupus originates. Inspect the angle between the pinna and the mastoid, and the interstice between the lobule and the skin of the cheek, for intertrigo. Eczema may be seen on the surface of the auricle and at the orifice, as well as inside the meatus. (See p. 440.) Note the occurrence of cysts, tumours, ulcers, etc.

The External Auditory Meatus.—Note malformations, collapse of the anterior or posterior walls, narrowing or stenosis of the calibre of the canal, inflammations of the lining, furuncles or boils, seborrhœa, impacted cerumen, débris, and, lastly, discharges. Before wiping away a discharge, observe its colour, quantity, and adhesiveness. Note whether it is serous, sanguineous, muco-purulent, or purulent.

Note whether moving the pinna to open the meatus causes pain.

In order to open the meatus in infants under two years of age, pull the auricle downwards and backwards; in children, between two years and five years, pull it directly backwards; and in older children and adults, pull it upwards and backwards.

The Mastoid Region.—Examine the prominence of the mastoid process, noting any swelling of the process or alteration in colour of the skin over it.

The parotid region and the parts about the angle of the jaw sometimes become infected in certain aural diseases; note, therefore, any unusual fullness or tender areas in these regions. The same applies to the sterno-mastoid muscle, and to the scalp behind the mastoid process.

Having completed the examination so far, take then an **aural speculum** of a size suitable for the canal under observation. For ordinary inspection purposes a circular white metal speculum with a well-expanded bell, and not too long a tube, is recommended. (Fig. 146.) (Urban Pritchard's pattern.)

A serviceable emergency speculum can be made of writing-paper twisted into a cone.

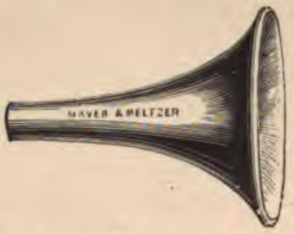

FIG. 146.—Urban Pritchard's Aural Speculum.

Having sterilized and warmed the speculum, draw gently on the pinna so as to open and straighten the meatus, and insert the speculum with a rotary movement. Remember the normal directions of the external meatus. First, it passes inwards, upwards, and slightly backwards; then, at the junction of the membranous with the osseous meatus, the canal bends, to run inwards, forwards, and a little downwards. Pulling the pinna upwards and backwards, therefore, tends to obliterate the angle and to convert a bent into a straight tube. The point of flexion is also the narrowest portion of the meatus, and is known as the isthmus. It is unnecessary to insert the speculum further than the isthmus, and, indeed to do so proves very painful. In infants the external meatus is very short, and the membrana tympani is situated very obliquely, facing almost directly downwards. At this age, it is frequently necessary to put the patient under a general anæsthetic in order to make a satisfactory examination of the ear.

When the end of the speculum has reached the isthmus in an unobstructed meatus, tilt or turn the head in various directions, and the membrane will come into view.

In passing the speculum, note any abnormality in the direction, depth, and calibre of the canal; note the presence of cerumen, débris, discharges—serum, blood or pus. If these interfere with a proper inspection of the membrane, they must be gently wiped away. Note, also, the presence of tender or inflamed spots near the orifice of the meatus, and the colour of the epidermal lining of the osseous meatus, which, usually pale pink, may become dusky-red in suppurations of the middle ear, etc. A general narrowing of the canal is commonly due to diffuse external otitis, secondary to long-standing middle-ear suppuration. A localized bulging of the walls of the canal near the orifice is usually furuncular, and when affecting the postero-superior quadrant of the deeper meatal wall is generally secondary to mastoid-antrum inflammation (see later). Exostoses, polypi, and granulations should be noted.

Next transfer your attention to the **membrana tympani,** which appears as semi-translucent, pearly-white, tense membrane blocking the inner end of the external auditory meatus. Much experience and practice are required before one can become confident of recognizing the various landmarks, even in normal ears. Remember that the membrane does not face directly outwards, so that the observer can look straight down on it, but that it is inclined more or less obliquely outwards, downwards and forwards ; we thus obtain only a very slanting view of the surface. As a result of this position, the posterior wall of the meatus passes into the posterior margin of the membrane insensibly as far as direction is concerned, and when the membrane is inflamed and of the same colour as the meatal wall it is often very difficult to tell where the one structure ends and the other begins. On the other hand, the anterior margin of the membrane, forming, as it does, an acute angle with the anterior meatal wall, is sharply defined. Sometimes a sharper bend than usual of

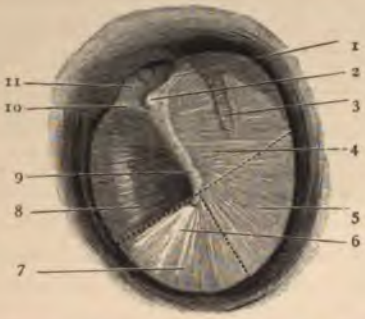

FIG. 147.—Left Membrana Tympani, from without (from Cunningham's Text-book of Anatomy).

(1) Posterior fold. (2) Short process of malleus. (3) Long process of incus. (4) Postero-superior quadrant. (5) Postero-inferior quadrant. (6) Cone of light. (7) Antero-inferior quadrant. (8) Antero-superior quadrant. (9) Malleus handle. (10) Anterior fold. (11) Membrana flaccida.

the meatus prohibits the insertion of the speculum far enough to afford us a view of the anterior edge of the membrane.

By an examination of many normal cases familiarize yourself with the landmarks of the membrana tympani. (Fig. 147.) These are—(1) the handle (manubrium) of the malleus, running downwards and backwards, to (2) its tip, the umbo, which lies rather below the centre point of the membrane, and from which the conical light-reflex passes downwards and forwards. Just in front of and above the upper end of the manubrium of the malleus is a tiny nipple-like projection, formed by (4) the short process of the malleus. Arching forward and backward from the short process are (5) two curved lines which separate the membrana vibrans below from the (6) membrana flaccida (or Shrapnell's membrane) above. The latter is duller and warmer in colour than the former. Observe the condition of the membranæ vibrans and flaccida. Note their colour, apparent thickness, lustre and transparency. Note the existence of calcareous deposits—dense white islets, generally situate in the posterior segment of the membrane, but sometimes forming an arc round the umbo, with the concavity upwards—the *arcus senilis* of the membrana tympani. Note the presence of *scars*, which appear as thinnings or thickenings of small areas of the membrane. Sometimes one can see through them, as through a window, into the middle ear; at other times they render the membrane very opaque. Cicatrization, when extensive, may so distort the membrane that it may be impossible to recognize the landmarks. As a rule, however, the short process and the handle of the malleus are recognizable, if they are present; but suppurative disease, of which this scarring is the result, may have destroyed the ossicles. When more or less of the membrane has perished as a result of suppuration, or some other destructive process, it will show *perforations*. Note their site, shape, and whether single or multiple. (See p. 485.)

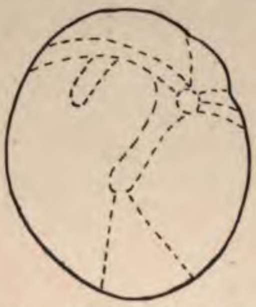

FIG. 148.—Outline of Membrana Tympani. Compare Fig. 149.

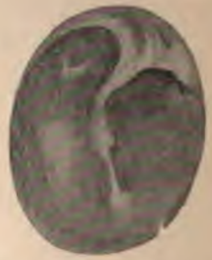

FIG. 149.—Retracted Tympanic Membrane.

Observe whether the membrane is retracted or bulging as a whole or in part. *Retraction* is manifested by a general loss of obliquity on the part of the membrane, the prominent upper and posterior segments receding into the tympanic cavity, and bringing the other parts into more direct view. Thus, the membrane looks wider than normal, because we can see more of the anterior segment. The handle of the malleus having been drawn in along with the sunken membrane, it assumes a position approximating to the horizontal, and as it is seen " end on," the foreshortening gives us the impression that the manubrium is shorter than it should be. (Fig. 149.) This displacement inward may be so considerable as to bring the tip of the handle into contact with the inner tympanic wall. The alteration in the disposition of the plane of the membrane produces also a displacement, distortion, or disappearance of the conical light-reflex in the lower segment. Finally, in extreme cases the circular margin of the tympanic ring is seen standing out around the indrawn membrane.

Adhesions may prevent the handle of the malleus from accompanying the membrane in retraction. In that case, no foreshortening is seen, but the segments of the membrane, in front of and behind the malleus handle, are sunk ; the prominence of the short process of the malleus is very much exaggerated ; the margin of the tympanic ring is too evident ; and, lastly, the various intratympanic structures are more or less visible. The typical picture is seen when, in front of and behind the prominent ridge of the handle of the malleus, two distinct hollows present themselves, and the membrane, like a tightly-drawn veil, stretches over the intratympanic structures, the most conspicuous of all being the short process and manubrium of the malleus.

It is essential to remember the relations of the membrana tympani to the contents of the middle ear. The main mass of the chain of ossicles occupies the attic, or upper chamber, of the tympanum, which lies on an altogether higher plane than the meatus (Fig. 150) ; so that when we require to probe the ossicles in the attic through a perforation, say, in Shrapnell's membrane, the extremity of the probe must be bent at right angles with the shaft in order to pass up into this cavity. The membrana tympani may be conveniently divided into four quadrants or segments by a line drawn through the handle of the malleus and extended downwards to the lower periphery of the membrane intersected at right angles by an equatorial line drawn through or a little above the umbo. The four quadrants thus formed are called the postero-superior, postero-inferior, antero-superior, and antero-inferior segments of the membrane. Of these the postero-superior is the most important. Underneath it is the long process of the incus, in a position roughly parallel with the direction of the manubrium of the malleus, but of course lying deeper, as well as higher, in the tympanum. At the end of the long process of the incus is its articulation with the head of the stapes, and at times this, together with the posterior crus of the stapes, passing upwards and inwards to the footplate in the fenestra ovalis, can be seen through the upper corner of the postero-superior quadrant of the membrane. The stapedius tendon passes back from the head of the stapes. The position of the long process of the incus and the head of the stapes varies considerably in different cases. When below or on a level with the short process of the malleus the incudo-stapedial articulation will lie directly under the postero-superior quadrant of the membrane ; but it not infrequently happens that the long process of the incus does not descend low enough in the tympanum for the incudo-stapedial joint to be visible below the superior margin of the osseous tympanic ring. At other times the long process

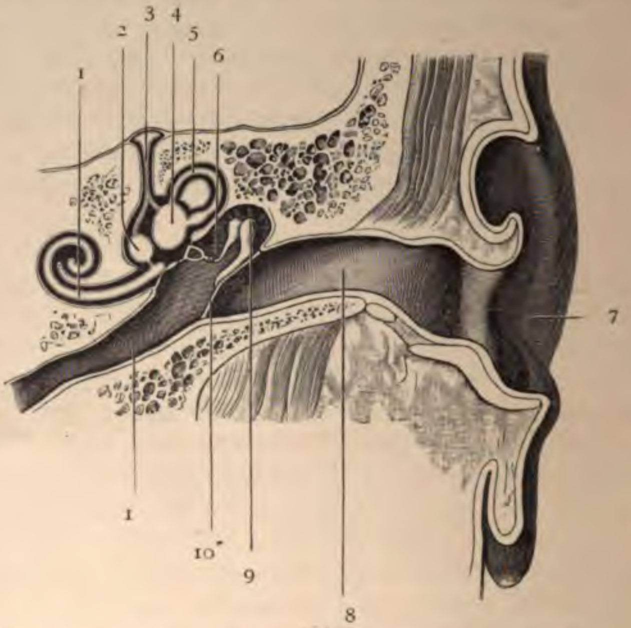

FIG. 150.—The Organ of Hearing from the front—Diagrammatic (Cunningham's Textbook of Anatomy).

(1) Cochlea. (2) Saccule. (3) Ductus endolymphaticus and saccus. (4) Utricle. (5) Semicircular canal. (6) Tympanic cavity with chain of ossicles,—malleus, incus, and stapes. (7) Pinna. (8) External auditory meatus. (9) Recessus epitympanicus or attic. (10) Membrana tympani. (11) Eustachian tube.

of the incus is concealed by the posterior margin of the tympanic ring, and will only come into view if the patient's face is turned towards the examiner, so as to bring the back parts of the tympanum into the line of inspection. These structures may be visible when the membrana tympani is very thin and transparent, when it is much retracted, or when there is a large perforation.

On the internal medial tympanic wall, occupying a site corresponding to the lower portion of the same quadrant (the postero-superior), is the fenestra rotunda, the plane of which looks downwards and backwards. This also varies in position. Sometimes it lies altogether lower than the inferior margin of the annulus tympanicus. Above and in front of the round window is the promontory, a convex rounded projection of the inner tympanic wall, marking the first spiral of the cochlea.

Arching forward across the upper hemisphere of the membrane, and occasionally visible as a white streak passing between the handle of the malleus and the long process of the incus, is the chorda tympani nerve.

The aqueductus Fallopii, carrying the facial nerve on the inner tympanic wall, lies well above the superior margin of the membrane, and cannot be seen in ordinary cases.

The postero-inferior quadrant overlies part of the fenestra rotunda, and the antero-superior corresponds to the opening into the tympanic cavity of the Eustachian tube.

In inflammatory disease of the middle ear examine carefully the postero-superior quadrant of the membrane, together with the adjacent part of the meatus; for here mastoid antrum inflammation is often revealed in the bulging of the membrane, and in depression or "sagging" of the meatus.

Examine carefully the membrana flaccida also, which gives access to the attic and ossicles, and to Prussak's space, a pocket formed by folds of mucous membrane lying between the body of malleus and the outer wall of the attic and tympanum. This is a common situation for circumscribed purulent disease. Perforations in the membrana flaccida are often small and inconspicuous, but they are, nevertheless, of great importance, since they indicate suppurative disease, past or present, affecting the attic, and perhaps the ossicles. In cases of suppuration of the middle ear with perforation, an examination by means of a fine probe, most delicately conducted, as the meatus is as exquisitely tender as the cornea, may be of great service in forming a diagnosis since we are thereby enabled to ascertain the condition of the ossicles and bony walls of the tympanum (See Fig. 65.) For details on these and other points, the reader is referred to the chapter on Affections of the Ear.

When we have thoroughly inspected the meatus, membrane, etc., by means of the ordinary speculum, the next step to be taken is an examination by means of **Siegle's pneumatic speculum,*** by which the membrane is inspected through a lens, and its mobility and that of the chain of ossicles tested. It is specially useful for revealing inconspicuous scars, small perforations, and scanty secretion.

Method.—Thoroughly dry and clean the meatus by mopping with a cotton-wool swab. Apply some dry soap to the inner surface of the lens on the Siegle speculum, and polish with a dry towel, to prevent clouding by the condensation of moisture. Then insert the speculum in the usual way, and examine the membrane carefully through the lens. Take note of scars, perforations, the tympanic structures visible, granulations, and any points or areas where pulsation may be seen. Then, with the orifice of the speculum fitting the meatus more or less closely according to your desire, and with the eye fixed on the membrane, alternately compress and relax the rubber inflating-ball, at first gently, and later, if necessary, more forcibly, so as to impart an in-and-out flapping movement to the membrane. By doing this, the amount of mobility of the membrane and ossicles can be estimated, and if there is any secretion, it can be sucked out of the middle ear. With regard to mobility, note whether the membrane is immovable, normally

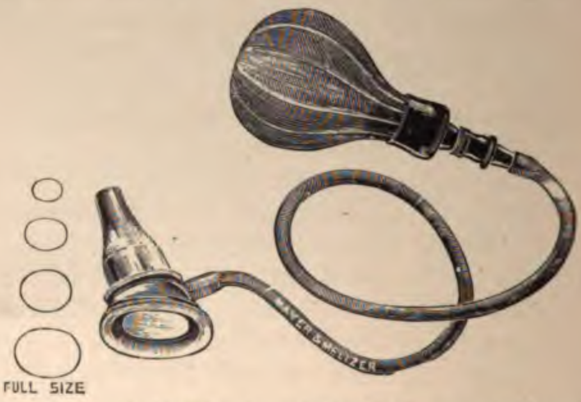

FIG. 151.—Peter's Modification of Siegle's Pneumatic Speculum.

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* Care should be taken in purchasing Siegle's Speculum to see that the lens is set at an angle of 45 degrees to the axis of the instrument; otherwise the light reflex will effectually blot out all view of the membrane.

movable, or thin and loose; whether the malleus handle accompanies the membrane in its excursions or not; whether any scars or atrophied areas move more or less than the healthy parts of the membrane; and whether there seem to be any adhesions limiting the movements of the membrane or malleus by binding them down to the inner wall. If there is secretion, note its character, and from what part of the membrane it seems to be coming.

Inflation of the tympanic cavity through the Eustachian tube by (*a*) Valsalva's method, (*b*) Politzer's method, or (*c*) by means of the Eustachian catheter, gives us information regarding (1) the patency or otherwise of the Eustachian tube; (2) the contents of the tympanum; (3) the state of tension of the membrana tympani; and (4) the presence or absence of perforations in the membrane.

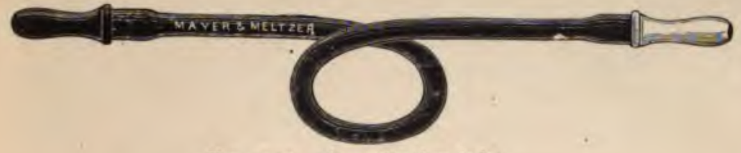

FIG. 152.—Auscultation-tube.

(*a*) **Valsalva's Method.**—Instruct the patient to pinch the nostrils tightly between his fingers and thumb, to close the mouth and by making a sudden and forcible expiration, to force air into the closed nostrils, and up the Eustachian tube, so as to inflate the tympanum and drive out the membrane. If, at the same time, we have the membrane under inspection, it is possible to watch it during inflation; or we may listen to the "impact sound" by using the auscultation-tube—a long rubber tube fitted at one end with a black nozzle, for insertion into the patient's ear, and at the other with a white nozzle, for insertion into the surgeon's ear. (Fig 152.)

Valsalva's method is seldom used nowadays.

(*b*) **Politzer's Method,** by Politzer's bag. The writer prefers a small bag, which can be easily grasped, provided with a long flexible rubber tube having a wide lumen, and a conical vulcanite or solid rubber nozzle, for insertion into the patient's nostril. (Figs. 153 and 154.)

Method.—Seat the patient facing you. Fix up the auscultation-tube. Gently insert the nozzle of the Politzer bag into the nostril of the same side as the ear in which your auscultation-tube is placed. Compress the sides of the nostrils against the

Politzer nozzle by pinching them tightly together with the forefinger and thumb of your left hand. Then, while you listen carefully, compress the bag with your right hand, at first *quite gently*, later, if necessary, more forcibly and suddenly. Then, if the Eustachian tube is patent, you will hear the click or snap of the membrane quite distinctly. (Familiarize yourself, by constant practice, with the different sounds heard in health and disease.) After one ear has been inflated, transfer your auscultation-tube to the other ear and listen to it.

Sometimes, instead of asking the patient to blow out his cheeks, we make him hold some water in his mouth, and the bag is compressed simultaneously with the passing of the water into the gullet.

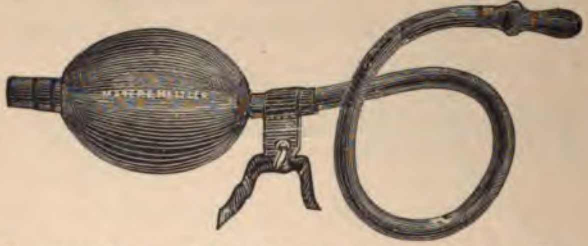

FIG. 153.—Lennox Browne's Modified Politzer Bag.

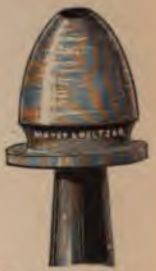

FIG. 154.—Nozzle for same.

In infants, inflation may be successfully performed when they are crying.

Auscultation Sounds in the Tympanum—*Normal.*—On inflating a normal tympanum, two sounds are heard: (1) a soft blowing, caused by the air passing through the Eustachian tube; and (2) the impact sound—a flapping outwards of the tense membrane, in consequence of the sudden elevation in intratympanic pressure. In a normal ear, the impact sound is faint. When the membrane is *indrawn*, the impact sound is exaggerated. When the membrane is *relaxed*, not only is the impact sound exaggerated, but often a second and fainter snap or click, like an echo of the first, may also be heard, produced by the membrane slipping back to its usual position, when the tympanic pressure is lowered again.

When there is *fluid in the tympanic cavity* the sounds vary according as the fluid is (*a*) permeable, or (*b*) impermeable by air.

(*a*) When the fluid is permeable, a rough bruit replaces the blowing Eustachian sound, and this is followed by moist crackling râles as the air bubbles through the fluid. If the fluid is confined to the attic, out of the course taken by the inflating air, no crackling will be heard.

(*b*) If the tympanic cavity is quite full of fluid, the air, rushing up the Eustachian tube, may compress it *en masse* without passing through it ; in that case, a dull and low-pitched impact sound will be heard, unaccompanied by crackling.

Perforation Sounds.—When a perforation in the membrane is not shut off from the Eustachian tube by adhesions, a whistling or blowing sound is produced on inflation by the escape of air through the opening. The pitch of the sound is high or low, according as the perforation is small or large.

All these sounds just described may be reduced or absent in cases where adhesions exist. The presence of adhesions may be suspected when, the Eustachian tube being patent, inflation is accompanied by neither crackling nor the impact sound.

Tubal Sounds are more readily obtained and differentiated when inflating with the Eustachian catheter than by Politzeration, but, for the sake of convenience, they will be considered here. The sounds emanating from the Eustachian tube vary with its diameter, its patency, the condition of its walls, and the presence or absence of moisture. When the tube is wide and clear of obstruction, the air rushes freely through it, and is heard as a blowing sound of a low pitch. When the tube is blocked, the sounds produced often give the listener some information as to the nature of the obstruction.

(1) In the case of a *plug of secretion*, repeated compression of the bag is necessary before the obstruction gives way. At first a harsh and more or less distant sound is heard, and this is gradually replaced, as the obstruction is overcome, by the typical near blowing sound.

(2) When there is swelling of the Eustachian walls themselves—e.g., from catarrh—the sounds produced depend upon whether this obstruction is permeable or not. When permeable, a harsh sound is heard at first, and persists unaltered. When impermeable, we cannot hear any tubal sound at all, or, at the most, only a distant whistling or faint blowing. If fluid is present in the tube, crackling râles may accompany the blowing sounds.

Note carefully, when you listen to an ear being inflated, whether the sounds you hear are near or distant. The most distant are those produced in or near the pharynx. Less distant seem those which proceed from the tube, while tympanic sounds appear so startlingly close that they may seem to the listener to be produced in his own ear.

Pharyngeal Sounds are, of course, of no diagnostic value in aural disease, but we must be able to recognize them, lest we confuse them with the true tubal and tympanic inflation phenomena.

When the Politzer bag is in the act of being compressed, a loud rough sound is frequently heard coming from the throat. This is due to the rush of air overcoming the resistance of the soft palate as it lies in contact with the posterior pharyngeal wall.

When the catheter is being used for inflation, a rough, harsh sound is produced if the point of the instrument is in contact with the pharyngeal wall in front of or behind the orifice of the Eustachian tube, and this sound will continue until the catheter is properly inserted into the tube.

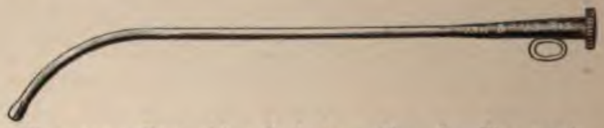

FIG. 155.—Eustachian Catheter. (See also Fig. 157.)

Catheterization of the Eustachian tube. The Eustachian catheter should be made of silver (Fig. 155) or other ductile metal. The pharyngeal end is curved more or less, according to the case, and the outer end should be well expanded and provided with a single ring to indicate the direction of the pharyngeal curve and point. The Eustachian catheter, when in position, should not project more than ½ inch from the anterior nares. A small rubber bag, valved, and provided with a pointed nozzle to fit accurately the outer end of the catheter, is necessary for inflation. (Fig. 156.)

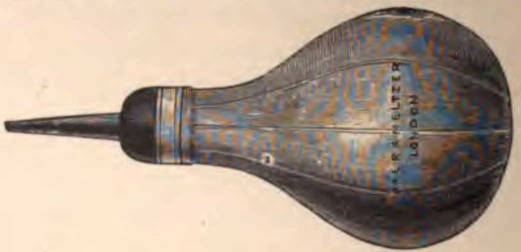

FIG. 156.—Eustachian Inflating-bag.

To pass the Eustachian Catheter, sit face to face with the patient, with the diagnostic tube in position. Under the guidance of the nasal speculum and your forehead light, pass the catheter, with the point downwards, along the floor of the nostril until you feel the convexity of the curve touch the posterior pharyngeal wall. Remove the nasal speculum; then, with your left hand, draw the catheter slowly towards you for about ¼ inch, at the same time rotating the instrument until the ring, corresponding to the tip, is pointing upwards and outwards towards the ear you wish to inflate. This will generally bring the tip into contact with the posterior lip of the Eustachian orifice, and a little more drawing forward, while at the same time you carry the outer end slightly towards the middle line, will, as a rule, suffice to engage the catheter well in the Eustachian tube (Fig. 157). Next, supporting the catheter in this position with the thumb and fingers of the left hand, insert the nozzle of the inflating-bag in the outer end of the catheter, gently compress the bag, and listen carefully to the sounds produced.

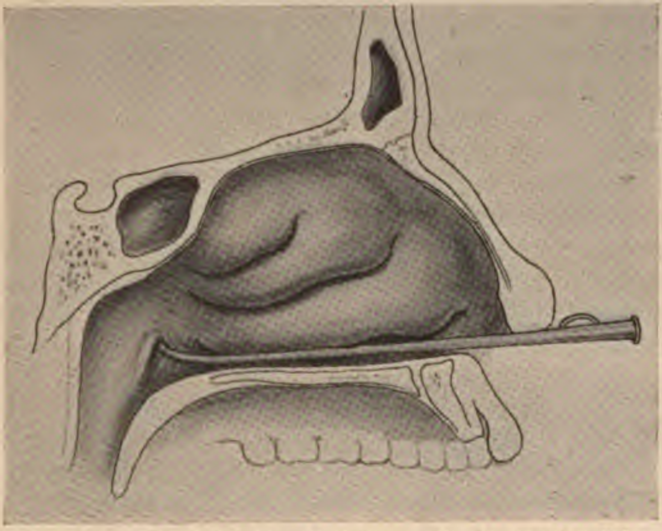

FIG. 157.—The Eustachian Catheter in the Eustachian Tube.

The position of the tubal orifice relative to the floor of the nose varies considerably in different people. In some, the ring and the tip of the catheter, when it is in position, will be found to lie in a direction pointing towards the top of the pinna of the ear to be inflated; in others they will be quite horizontal. Thus, while the foregoing directions hold good for most cases, it will be often found necessary to depart from them, since the Eustachian orifice may be met with at any point in an arc of about 60 degrees.

Failure to engage the point of the catheter in the tubal orifice will be evident when absolute obstruction is presented to the compression of the inflating-bag, or when the rough pharyngeal sound described above is heard.

On the other hand, that we have been successful in passing the catheter will be obvious when, on inflation, tubal or tympanic sounds are audible.

If after several attempts to engage the point in the orifice you continue to meet with failure, withdraw the instrument entirely, alter the pharyngeal curve one way or another, try with a longer or a wider instrument, or pass the catheter under endoscopic inspection. (See below, p. 435.)

Another Method of catheterizing the Eustachian tube sometimes succeeds when that just described fails. This consists in rotating the catheter after it has passed the nose, so that the point is turned inwards, at the same time drawing the instrument forward, so that the convexity of the curve catches on the posterior edge of the nasal septum. The catheter, held at this level, is then rotated until the point is directed upwards and outwards, when it will arrive at or opposite to the Eustachian orifice.

Difficulties.—Many obstacles may hinder or prevent catheterization, but they can frequently be avoided or overcome. Most of these are due to septal deviations or spurs, occluding the nostril more or less completely. Sometimes we get over this difficulty by slipping the catheter under the spur, straightening the curve of the instrument in order to do so. If this proves unavailing, the catheter may be made to pass the obstruction by inserting the instrument into the nose with the curve downwards, rotating it into the proper position after the naso-pharynx has been reached. Or, again, it may be inserted above the obstruction, in which case it will lie at a higher level than the floor of the nose, so that, in order to bring the point into the Eustachian canal, it will be necessary to depress the tip by raising the outer end. Sometimes it is possible, after thus inserting the catheter over the obstruction, to insinuate the shaft of the instrument between the septum and the side of the nostril to its proper place on the floor.

Sometimes, in spite of all endeavours, it will be found impossible to pass the catheter through an obstructed nostril. In that event, the Eustachian canal may be reached through the other nostril by a catheter with a long curve and tip; or a suitably constructed tube passed through the mouth and up behind the fauces may be tried. In most instances, however, it will be found possible to catheterize through the nose in the usual way. If not, then the obstruction, by deflected septum, spur, and what not, being so marked as to prevent the passage of the cannula, should receive surgical treatment, not only in order to enable us to use the catheter as a method of treating the deafness, but also in order to relieve the nasal obstruction which, in cases such as these, is very considerable.

All the manipulations necessary for effectual catheterization should be conducted with great gentleness and delicacy, so as to avoid wounding the mucous membranes of the nose and nasopharynx. When the patient is very sensitive, the application of a 10 per cent. solution of cocaine to the nasal mucous membrane will render the act quite painless.

Catheterization under Endoscopic Inspection.—In cases where the catheter can be passed through the nose, but where it does not seem to engage in the orifice of the Eustachian tube, the nasopharyngoscope, or nasal endoscope, should be employed, and the point of the catheter passed under its guidance into the Eustachian orifice. (Sec Plate II. Fig. 2.)

If, while it is in this position, and inflation is practised, the air does not reach the tympanum, then obstruction of the Eustachian tube may be diagnosed.

CHAPTER XII

AFFECTIONS OF THE EAR.—THE AURICLE.

Malformations.—(1) **Congenital Malformations** of the auricle occur in considerable variety. The lobule may be cleft, abnormally large, or entirely absent.

In the region of the tragus, the auricular appendages are found, relics of the cartilage of the first branchial arch, and in front of the upper part of the pinna the curious fistula congenita auris is sometimes seen.

Treatment.—The treatment of these deformities is surgical. Abnormally large lobules should be split, the fibrous tissue and fat removed, enough being left to supply the foundation of the new lobule, which is covered all round with skin from the old, redundant parts being pared off.

The cleft lobule may be cured by paring the margins and apex of the cleft, and bringing the raw edges together with horsehair sutures.

Auricular appendages, consisting of clumps of contorted cartilage in the cheek in front of the auricle, necessitate a deeper dissection than appears at first sight. There is no need to remove the whole mass, however, only so much being excised as is necessary to remedy the deformity.

Congenital Deficiency, or Absence of the Auricle, is a much more serious deformity than those just mentioned, seeing that in addition to the auricular defects, the external auditory meatus, the tympanum, and the labyrinth are also involved.

Not infrequently also there is present congenital facial paralysis and hemiatrophy of the face on the same side.

Symptoms.—The patient is brought on account of the auricular deformity, and the associated deafness is frequently not remarked.

We may interpolate here the remark that severe unilateral deafness of congenital origin, or of a very long standing in children, is difficult to diagnose, if the other ear is healthy, because the normal ear supplies the place of the deaf ear so surprisingly well. The child will, for example, quite frequently hear the whisper at normal ranges, when the sound ear is stopped with the finger.

On examination, the auricle is seen to be more or less deficient —perhaps only a few shapeless nodules of skin-covered cartilage being present.

The speculum shows the external meatus to be occluded, its place being represented by a hard fibrous cord leading inwards.

Tests, when they can be carried out, will show that there is no hearing whatever on the affected side.

Treatment.—No treatment is of any avail. Attempts to fashion a new external meatus by operation should not be undertaken unless there is indubitable evidence that the cochlea on the affected side is active.

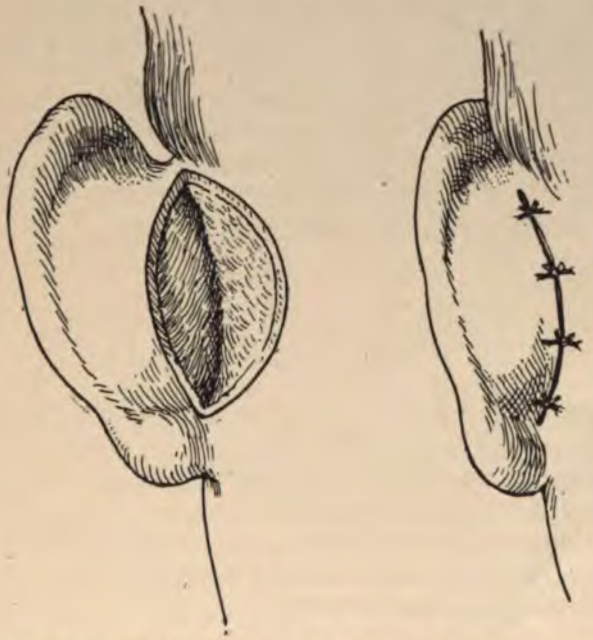

FIG. 158.—Ear. Plastic Operation for Unduly Prominent Auricles.

Unduly Prominent Auricles projecting out from the side of the head constitute a deformity which the ear-surgeon may be asked to remedy.

Operation.—A crescentic incision tailing off towards the lower end (see Fig. 158) is made over the back of the auricle, and a corresponding incision is made over the mastoid region. The skin surrounded by those incisions is entirely dissected off, leaving a continuous raw surface on mastoid and auricle.

We come now to the indispensable part of the operation. Projecting into the front of the auricle, and bordering the concha is a sharp lamina of cartilage, which is known as the crus of the antihelix. (Fig 159.) In order to get the auricle to lie back close to the mastoid region, the continuity of this lamina of cartilage must be interrupted. To do so it is incised from the wound on the posterior aspect of the auricle, dissected free of the skin on its anterior aspect, care being taken not to button-hole the skin, and a suitable piece resected.

The auricle is then flattened backwards so as to bring the raw surfaces together where they are retained by sutures through the edges of the wound now in contact with each other. (Fig. 158.)

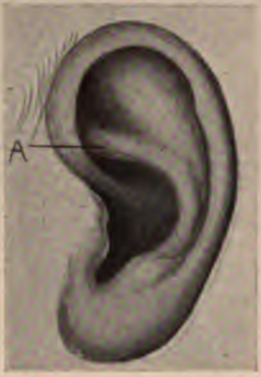

FIG. 159.—Operation for Protuberant Auricles. *A* Crus of the Antihelix, resected in the operation.

INJURIES TO THE AURICLE

The commonest injuries of the auricles are blows received in boxing and gunshot and shell-wounds producing lacerations and contusions. The latter may cause hæmatoma auris, and both types may be followed by perichondritis.

Local Treatment.—Lacerations, after being cleansed of gross material impurities, and rendered as aseptic as possible, are united by sutures passed through skin, the auricle being reconstructed as far as possible. Thereafter fomentations by boric acid lint are generally necessary, as there is considerable inflammatory reaction, which may pass on to perichondritis (q.v.).

Hæmatoma Auris is the result of a contusion of the auricle. It consists in a subperichondrial hæmorrhage, which separates the perichondrium from the underlying cartilage. In civil life, hæmatoma is common in boxers, and occurs spontaneously, it is said, in the insane. The swelling of a hæmatoma is livid, roundish, and tense, and it may involve any part of the auricle, save the lobule. The absorption of the effused blood is generally accompanied by a certain amount of quiet perichondritis, whereby more or less permanent thickening of the tissues of the auricle at the site of the injury is produced. If the injury is frequently repeated, the whole pinna becomes dense, infiltrated, and somewhat nodular, a condition known as "prize-fighter's" or "boxer's" ear.

The *Treatment* of hæmatoma auris is surgical if seen early. The tumour should be incised under strict asepsis, the contained fluid and blood-clot evacuated, and the incision closed with fine sutures passing through skin only, not cartilage. A thin gauze and collodion dressing should be carefully applied to the auricle, and the whole covered with absorbent wool and gauze, kept in position by a firmly-applied bandage. This outer dressing must be removed daily for inspection of the wound and site of injury, as the cavity of the blood-cyst is very liable to refill with serum, in which case a stitch must be removed, and the cavity emptied by pressure.

Perichondritis of the Auricle is septic in origin, and generally follows an injury causing laceration or hæmatoma, which becomes infected. It is an occasional manifestation of sepsis after the radical mastoid operation when the auricle has been incised by the surgeon's knife cutting the meatal flap. Any of the pyogenic organisms may be responsible for the disease, but the bacillus pyocyaneus bears a particularly evil reputation in this respect.

Symptoms.—The auricle becomes burning, throbbing, and painful; it is tender to the touch; and as a whole, swollen and thickened, coming to look larger and bulkier than normal. In the severer cases, subperichondrial collections of thin pus form, and there is a certain amount of pyrexia and constitutional disturbance. Under simple treatment, the disease tends to complete recovery without deformity, but in the severer cases the auricular cartilage is damaged by the abscess formation, and, usually without massive necrosis but rather by a molecular disintegration and absorption, undergoes considerable shrinking and malformation.

The disease lasts for several weeks, even in its simplest forms.

Treatment.—The prevention of this tedious infection in injuries, whether traumatic or operative, is secured by strict asepsis. But this desideratum is sometimes impossible to obtain, as in the radical mastoid operation.

After the disease has set in, the ear should be treated with warm boric acid fomentations. Fluctuating swellings should be incised whenever found.

Frost-bite.—The isolated position of the auricle, and its deficiency in protective subcutaneous fat, render it, when exposed to severe cold, very liable to frost-bite. The signs are loss of the feeling of tingling or cold, from anæsthesia of the part, with stiffness when touched, and very noticeable pallor. If neglected, perichondritis and even gangrene result.

Treatment.—The immediate treatment consists in gentle assiduous friction with snow, if possible, or with the bare hand. The immediate application of heat is dangerous.

SKIN AFFECTIONS OF THE EXTERNAL EAR

Intertrigo is found at the junction of the skin of the pinna with that of the mastoid and temporal regions, and in the recess formed by the lobule with the cheek. It is common in unhealthy children and infants. Characterized by serous exudation with redness and rawness of the opposing cutaneous surfaces, it is distinguished from eczema by the absence of infiltration in the affected areas.

Treatment.—In the simpler cases, dusting with zinc oxide and lycopodium powder will lead to cure. If not, it should be treated as eczema (q.v.). The general health should be attended to.

Acute Eczema, while it may result from the extension of an intertrigo, is, in most cases, secondary to suppuration of the middle ear, and it often forms a troublesome complication of the acute variety of that disease in unhealthy children. In these cases the meatus is chiefly affected, in which situation the subcuticular thickening and infiltration may, by narrowing the meatal lumen, seriously incommode the treatment of the graver disease.

The *Symptoms* are those of acute eczema elsewhere—redness and swelling of the skin with exudation through raw surfaces and " chaps " of pinkish, irritating serum in drops. The disease is frequently bilateral, but in the septic type it remains usually limited to the meatus and auricle, and especially to the conchal surface of the latter. The acute variety accompanying acute middle ear suppuration, after the initial outburst, tends gradually to become less severe, and finally to die away, but, especially in neglected and dirty children, it may pass into an obstinate and intractable state with fibrous thickening of the skin.

Treatment.—For the acute eczema of middle ear suppuration, the meatus is treated with simple ungt. ac. boric. inserted round the walls, so as to protect them from the irritation of the antiseptic watery solutions used for the middle ear, and these last should, of course, be as mild as possible.

For the auricle, otherwise, nothing in these acute cases is better than liq. plumb. subacetat. dil. applied on clean cotton. Later, as the redness and inflammation subside, zinc oxide, calamine, and ichthyol may be used. And for the older and more obstinate cases, ungt. hydrarg. nit. mit. is useful.

Syphilitic Infiltration of the Auricles and Meatus.—A subcutaneous fibrosis, producing a local appearance resembling a mild elephantiasis, is an occasional manifestation of the syphilitic virus. The disease is symmetrical, and it is limited in distribution to the auricle and meatus. The thickening of the meatal walls causes stenosis of the canal, and produces deafness.

It is not quite clear whether this appearance is ever anything but syphilitic. The few cases I have seen yielded to nothing but anti-syphilitic remedies, and even to them it proved to be very obstinate.

(We mention it at this juncture because it closely resembles simple chronic eczema.)

Seborrhoic Eczema of the auricle is not uncommon. It produces scaliness of the anterior aspect of the pinna and of the meatal walls. It is usually found in association with the same disease elsewhere, and scaliness with the familiar eczematous reddening of the skin of the scalp is almost invariably present.

Treatment.—Water should be avoided, the ears being cleansed with salad oil. Perhaps the best local application is an ointment of salicylic acid, grs. 10 to 15, to the ounce of soft paraffin.

Impetigo Contagiosa is characterized as elsewhere by discrete, circular, raw patches, later becoming confluent, the exudation drying into curled, flaky, honey-coloured crusts. Its contagious nature is evidenced in the presence of patches elsewhere, generally about the roots of the nails in unhealthy children.

Treatment.—The crusts must, first of all, be thoroughly got rid of, and for this, bread and starch poultices may be employed.

The rawed surface is then treated with dilute ungt. hydrarg. ammon.

Impetigo is easy to cure, but difficult to eradicate, recurrences being frequent.

Lupus Vulgaris, when it attacks the auricle, usually does so by extension. As a primary disease, it is rare.

Lupus Erythematodes, on the other hand, manifests a preference for the auricle. Here it is marked by a red glazed skin surface with loose dry crusting, thinning of the skin, and also a degree of thinning and atrophy of the auricular cartilage, the finer mouldings and edges of which are blurred and smoothed out.

HERPES ZOSTER OTICUS

(We may most conveniently consider Herpes Zoster Oticus at this point.)

Etiology.—Herpes zoster is considered to be due to an inflammation of the posterior root ganglia of the spinal or cranial nerves. The disease does not, however, remain limited to the sensory elements It may extend to involve motor trunks, and then muscular paresis or paralysis is combined with the herpetic and par- or an-æsthetic phenomena.

Herpes of the auricle may be due to involvement of the posterior root ganglia of the upper cervical nerves, being implicated through the posterior auricular. Or, and this is the most important variety to the otologist, it may be due to herpetic disease attacking the geniculate ganglion of the facial nerve, in which case the auricular and facial herpes are combined with *facial paralysis.*

Indeed, there is some reason for the belief that most cases of facial paralysis, if not central or due to some form of tumour or to otitis media, are really herpetic in nature, and that the so-called " rheumatic " facial paralysis is non-existent. Whether this view of the matter is correct or not, there is no doubt whatever that herpetic facial paralysis is by no means an uncommon disease.

It is to be noted that in many, perhaps most, cases the herpetic disease is not limited to the facial nerve, but affects other individuals of the cranial nerves, most usually the vestibular and cochlear nerves. Indeed, the disturbance may be rather extensive.

Symptoms.—The first symptom is deep, burning pain in the ear, which is apt to be mistaken for the pain of acute suppuration. The herpes in its distribution corresponds to the sensory nerves attacked by the disease. Thus, it may be found on the auricle, in the meatus, on the membrane tympani, on the scalp, neck, side of the face, tongue, pharynx, tonsil, palate, forehead. On the other hand, it is often so slight and limited in distribution that it entirely escapes attention. The parts affected by the herpes become red and swollen, as if they were the seat of erysipelas. The external auditory meatus is closed by the swelling, and as some serous discharge exudes from the canal, the resemblance to an acute middle-ear suppuration with mastoiditis may be very close.

The facial paralysis is gradual in onset, becoming complete in the course of two or three days.

The vestibular symptoms—vertigo, nausea, vomiting, nystagmus—and those due to involvement of the cochlear nerve—tinnitus and deafness—appear in their turn, and there may also be found some oculo-motor paralysis. The motor paralysis may indeed extend as far as the larynx, the trapezius, and the sterno-mastoid.

The disease-phenomena are almost invariably limited to one side of the body, like herpes elsewhere.

The cerebro-spinal fluid shows lymphocytosis.

During the period of evolution the patient is depressed, miserable, and looks and feels very ill.

The herpes dries up and disappears in from a week to ten days. If incomplete, the facial weakness lasts for from two to four weeks only, but when it is complete, the paralysis does not begin to show any signs of recovery for from six months to a year, and the recovery may never be quite complete. In like wise, the acute vestibular phenomena disappear, but the ear remains irresponsive to the vestibular tests, and what is of more consequence, the deafness—which is of the " nerve-deafness " type—persists irremediably. The other paralyses disappear in time.

Diagnosis.—In its herpetic stage, the disease, as it affects the ear, is liable to be mistaken for acute suppuration. But the temperature in herpes is normal, the exudate in the canal is non-purulent, and the presence of the " corymbose clusters" of herpetic vesicles elsewhere, ought to be sufficient to settle any doubt that may arise. Facial paralysis without herpes, of course, appears sometimes in middle-ear disease. (See p. 593.)

In the pharynx, the herpetic spots are liable to be mistaken for lacunar tonsillitis. But their unilateral distribution, and their presence on the tongue and posterior pharyngeal wall should prevent mistakes being made.

Facial paralysis following a so-called "tonsillitis" is almost certainly herpetic in nature.

Treatment.—During the stage of invasion, which lasts from a week to ten days, the patient should be kept in bed. The herpetic vesicles may be dusted with zinc or starch powder, and if they break out in the mouth or pharynx, a mild antiseptic mouthwash and gargle should be prescribed.

During the period of development of paralyses, nothing can be done, but after the storm has subsided, the restoration of motor function may be aided by judicious electrical treatment, the faradic or galvanic current being used according as the muscles respond to the one or other.

CYSTS AND TUMOURS OF THE AURICLE

Sebaceous cysts are occasionally found on the auricle, usually in or near the lobule. They can be removed under local anæsthesia, the cyst being split from base to apex, the contents extruded, and the lining pulled out with forceps.

Fibroma occurs on the lobule often as a consequence of wearing ear-rings.

Warts or papillomata, and angiomata are occasionally met with.

Solid tumours are removed under local anæsthesia. Papillomata may be treated with silver nitrate, trichloracetic acid, and other caustics. Angiomata, if large, must be dissected out by incisions carried wide of the growth, save when they are of small size and amenable to local agents like diathermy, the constant current, or carbon dioxide snow.

CANCER OF THE EAR

Under this heading we shall discuss the whole question of ear-cancer.

Epithelioma.—*Pathology.*—The disease may start in the auricle, the meatus, or the middle ear spaces. Epithelioma of *the auricle* is commonest in old men. Beginning usually on the posterior aspect, necrosis of the cartilage is induced as it extends, and if left to grow unchecked, the whole auricle will be destroyed, leaving a vascular painful ulcer on the side of the head with raised serpiginous edges and exuberant granulations pouring out a copious fœtid discharge.

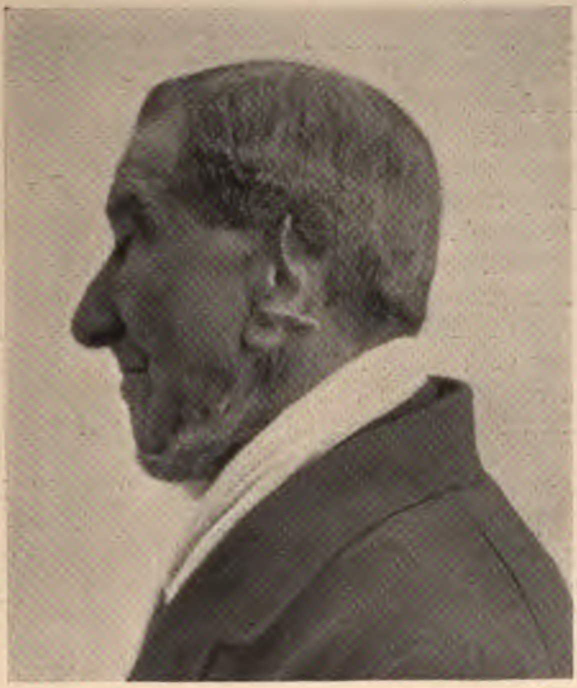

FIG. 160.—Epithelioma of the Auricle removed by the Diathermy Knife.

The glands attacked are, first, that on the mastoid process, and later, those of the deep carotid chain.

Epithelioma of the *deep meatus or middle ear* seems to bear a definite relationship to chronic suppuration, and one or two cases have been reported in which it seemed to originate in trauma resulting from a foreign body (Sir W. Milligan). In those deep regions, extensive destruction of the bone is rapidly produced, the facial nerve is attacked and destroyed, with consequent facial paralysis, and the dura is exposed and finally may be eroded.

When the growth starts in the meatus, it induces irritation with crust-formation, until an ulcer forms, which spreads out to the auricle, and in towards the middle-ear spaces.

A prominent symptom of ear cancer is the very severe pain.

Treatment.

Epithelioma of the Auricle should be treated by partial or complete amputation of the auricle, with removal of at least the mastoid gland. Raw surfaces left after amputation may be covered with grafts or the lobule may be split and reflected up to cover the denuded area.

The use of diathermy in place of the knife for this operation is recommended, care being taken to avoid destroying bone with the diathermy terminal. (Fig. 160.)

Epithelioma of the Meatus and Middle Ear necessitates the exposure of the disease through the mastoid, and the free removal of malignant granulations and bone. In an advanced case, very extensive removal of tissue may be necessary. Although several cases of freedom from early recurrence after operation have been reported, the chances are naturally not good, but the operation is very successful in removing the excruciating pain.

Sarcoma and **Endothelioma** of the **Temporal** bone are rare new growths. They form rapidly-growing tumours, frequently encapsulated, usually in the mastoid region, so that their removal through a mastoid opening is easily accomplished.

THE EXTERNAL AUDITORY MEATUS

FURUNCULOSIS (ACUTE CIRCUMSCRIBED EXTERNAL OTITIS)

The cause of furunculosis is a staphylococcus infection which in the case of the auditory meatus probably obtains an entrance from scratching with the finger. The cartilaginous meatus is the seat of the disease, and that for two reasons: firstly, because it is richly supplied with hair follicles; and, secondly, because it is well within reach of the patient's finger. Of this part of the canal, the inferior, posterior, and superior walls are more liable to attack than the anterior. Furuncles in the auditory meatus come in successive crops, just as they do when they appear on the skin elsewhere.

Symptoms and Signs.—After an initial feeling of more or less irritation and discomfort in the canal, lasting, as a rule, several days, pain, centring in the neighbourhood of the meatus, sets in, and gradually becomes very severe. There is also present pain and difficulty in moving the jaw and opening the mouth, so that mastication is interfered with. Headache, malaise, and sometimes slight fever, accompany the local symptoms. After a period of from one or two days to a week, a discharge of blood and pus appears from the meatus, and if the furuncle is solitary, relief from the pain follows ; but if, as is generally the case, one boil is accompanied or followed by several others, the pain continues intermittently till all have discharged. The tumefaction of the soft tissues of the meatus by the furunculous inflammation leads to narrowing, and ultimately to closure of the canal, with the usual signs of marked obstructive deafness, coupled with more or less tinnitus.

A history of severe pain in the ear, of sudden onset, intensified on opening the mouth or chewing, and associated with tenderness when the auricle is moved, should always lead us to suspect furuncle.

The value of the rule of making the first inspection of the ear without a speculum is particularly noticeable in these cases. As we have seen, the disease most frequently attacks the outer portion of the meatus, where the hair follicles are congregated, and if a speculum be used straight away, the instrument may be inserted past the tender spots near the orifice, and the actual lesions overlooked.

In furuncle of the anterior wall the tragus is often involved in the swelling, and the infective process may spread to the parotid gland, with which the anterior wall of the meatus is in direct relationship. Furuncle of the posterior wall is often accompanied by œdema and duskiness of the skin over the mastoid, and when this is associated with protrusion of the auricle as a result of the general inflammatory tumefaction, it may lead to the supposition that we are dealing with mastoid abscess secondary to middle-ear disease.

In the earliest stages the meatal wall may be so little altered from the normal that furuncle may never be suspected. In such cases, careful probing of the canal with a cotton-tipped probe will result in the discovery of one or more exquisitely tender areas. These mark the spot where the furuncles are forming.

In the latter stages, the tumefaction of the walls, and consequent narrowing of the lumen of the outer portion of the meatus are visible on simple inspection, and leave no doubt as to the diagnosis.

The condition of the deeper (osseous or membranous) portion of the canal depends upon the extent and duration of the furunculosis. For an effectual examination of this part of the canal, a speculum is, of course, necessary, and as it must be insinuated through the obstructed and inflamed canal, a speculum of the smallest bore should be employed. Once we have succeeded in passing the instrument through the stenosed part of the canal, the deeper portion will generally be seen to be little, if at all, altered from the normal. The wall, perhaps, may be a little more red than usual, but the membrana tympani is intact and of a healthy colour. Further, the insertion of the speculum past the stenosed portion of the canal will at once remove the deafness. If, however, a furuncular abscess has broken in such a way that the pus is discharged into the deeper canal, and there is complete obstruction at the orifice of the meatus, which is not relieved by this spontaneous evacuation—quite an ordinary occurrence, as furuncles develop in crops—then the deeper portion of the canal will be filled up with blood, pus, and débris, possibly under considerable pressure. As a result, the cutaneous layers of the meatus and of the membrana become sodden, macerated, and more or less septic, and the acute *circumscribed* external otitis is complicated by acute *diffuse* external otitis, affecting the deeper canal together with the membrane.

Indeed, the process may proceed a step further, for the pus may break through the softened tympanic membrane, and set up suppuration in the middle ear.

In severe cases such as these, the great pain and tenderness present may prevent the introduction of a speculum past the inflamed zone of the meatus, and we must be content to wait until proper surgical treatment has relieved the meatal furunculosis before we are able to obtain any information as to the state of the deeper parts.

Diagnosis.—It is necessary to distinguish furunculosis from that form of acute circumscribed external otitis of the bony meatus which occurs on the postero-superior segment of the meatal wall close up to the tympanic membrane, and which is due to an extension of the middle-ear suppuration to the mastoid antrum, leading either to an actual bulging into the lumen of the meatus of the thin anterior wall of that cavity, or, it may be, to sympathetic œdema and swelling of the lining of the meatus which covers the wall of the antrum. The following consideration will keep us from straying from the path of rectitude. In furunculosis the tenderness, redness, and swelling are found at or near the meatal orifice; while in bulging of the postero-superior meatal wall from antrum disease the canal is seen to be narrowed only in its deeper parts in the neighbourhood of the membrane.

When there is œdema of the mastoid from furuncle of the posterior wall, and especially when the auricle, as a whole, is abnormally prominent, it may be extremely difficult to decide whether the case is one simply of furuncle, or whether it is a case of furuncle combined with mastoid disease. In order to clear up doubt on this point, we make careful digital pressure over the mastoid, in such a way as not to move in the slightest the auricle or the cartilaginous meatus. If this pressure causes pain, mastoiditis is probably present; if it is painless, the disease is probably uncomplicated furunculosis.

Another sign of occasional value in the differential diagnosis is that when the mastoid œdema is due to mastoïditis the retro-auricular sulcus is deepened; when it is due to furunculosis, the sulcus is involved in the œdema, and is more or less obliterated. But this sign is at times misleading, and indeed it is the fact that the symptoms may be so equivocal that for some cases the diagnosis remains in doubt until the furunculosis is cured, or the mastoid is opened. And this is specially liable to be the case when, as sometimes happens, a furuncle of the postero-superior meatal wall has induced, by extension, the formation of a true abscess in the upper part of the mastoid region.

Treatment.—Under nitrous oxide anæsthesia, an incision is made through the most swollen part of the meatus down to the bone with a stout scalpel. If two furuncles seem to be present, two incisions are made. And then with a small sharp spoon or curette, the core and contents of the boil are scraped thoroughly out. A strip of sterilized gauze is inserted into the canal, and a hot fomentation is applied to the side of the head, to be removed in twenty-four hours.

This small operation may be employed at any stage of the disease, and its effect on the pain is immediate. Recurrences are best prevented by antiseptic drops, such as hydrarg. iodid. in aq. (1-1000) or, menthol, grs. iii. to the ounce of liquid paraffin instilled twice a day into the meatus.

For the great irritation of the canal, which is sometimes experienced during convalescence, Politzer recommends weak, white, precipitate ointment.

Some weeks after operation, an obstinate granulation, covering a small patch of bare bone, may persist in the canal. It gets well spontaneously.

Genuine abscess-formation over the mastoid process, or in front of the tragus, is easily drained through the meatal incision.

This treatment suffices to cure the active furuncle, and by removing the infection tends to prevent others developing. But the disease is sometimes obstinate, and in spite of all local measures one boil after another makes its appearance and the illness is prolonged for weeks and even months. In such cases a stock staphylococcus *vaccine* is often of value.

DIFFUSE EXTERNAL OTITIS

is acute or chronic.

As a rule, **Acute Diffuse External Otitis** supervenes upon the chronic disease (see later) in consequence of some superadded virulent septic infection. It may also be induced by the insertion of irritants; it may follow injuries, from infection of wounds not necessarily severe of themselves; and it may result from septic infection of a furunculous or epitheliomatous ulcer.

The inflammation varies in severity from simple dermatitis to phlegmonous or membranous inflammation. Gangrene has been known to occur, but it is very rare. The *symptoms* vary accordingly. In the severer cases, great pain, dusky swelling of the meatal walls and auricle, with the formation of vesicles, and the occurrence of a sanious discharge, accompany high fever and grave constitutional disturbance. In the milder cases, which are the more common, the symptoms resemble those of furuncle, but are less acute. Deafness, more or less, according to the degree of meatal obstruction, and tinnitus, are usual complaints, together with a certain amount of local pain and tenderness. On examination, the whole canal, including the osseous segment, is seen to be narrowed from tumefaction of its walls, and its lumen is occupied by softened epithelial débris. When this has been carefully wiped away, the walls can be scrutinized. Ulceration of the membrana or pouting granulations covering bare bone in the osseous canal may be found.

When granulations are seen in the neighbourhood of the membrane, it may be impossible to distinguish acute external otitis from purulent otitis media, with a perforation in the membrane, through which granulations are protruding. (See p. 487.)

Middle-ear suppuration may, of course, induce acute diffuse external otitis.

The *prognosis* of acute diffuse external otitis is favourable even in the severer cases.

Treatment.—In the earlier stages, the application of cold by means of Leiter's coils is at times serviceable in moderating the symptoms. Locally, the canal should be gently syringed out with weak lysol solution and powdered boric acid insufflated (Politzer).

Under the heading of **Chronic Diffuse External Otitis** is included a variety of conditions, all of which excite inflammation of the lining of the meatus. Thus it may arise from traumatism, from the introduction of irritating substances into the canal, from the presence of foreign bodies, from suppuration of the middle ear, or from vegetable parasites. In like manner, the extent of the inflammatory process ranges from simple eczema or seborrhœa of the dermal lining of the canal to chronic periostitis of the bony segment. As a result of long-standing chronic inflammation of the subcutaneous layers and of the periosteum permanent atresia of the canal may result.

Chronic Desquamative External Otitis, or Cholesteatoma of the Meatus is the name given to those cases, by no means uncommon, where the deeper part of the external auditory meatus is filled with foul, pultaceous débris, mingled with friable pieces of brownish or fawn-coloured membrane, of cholesteatomatous odour, the whole being contained in a membranous bag like the finger of a glove, of a white glistening appearance where it lies in contact with the meatal epidermis.

Pathology.—The disease is obviously a true cholesteatoma inasmuch as it is produced by the simultaneous desquamation of layers of dead epidermal cells, which, instead of drying up and disintegrating, after being shed, remain united in sheets or membranes. The gradual filling of the canal with this material leads in time to a dilatation of the deep bony meatus. The cause seems to be unknown.

Symptoms.—The patient usually complains of deafness, with a sense of fullness in the ear I have known it the cause ot extremely severe pain.

On examination, the material in the meatus is apt to be mistaken for impacted cerumen, but the probe will prove it to be soft, while the odour of cholesteatoma is characteristic.

It is also liable to be mistaken for cholesteatoma of the antro-tympanic cavities, and frequently it is impossible to arrive at a diagnosis until the canal is cleared.

Treatment.—Forcible syringing is recommended, but, as a matter of fact, it is generally impossible to eject the cholesteatomatous masses with a stream of water. Even solution of hydrogen peroxide is ineffective. The only resource is to remove the masses together with their lining membrane with aural forceps, spud, and blunt spoon, and as this process is at once very slow and very painful, much patience, and perhaps several sittings are required. Indeed, an anæsthetic may be advisable in some cases.

Once, however, the canal has been cleared of its contents, recurrence is easily prevented by keeping the canal dry, and by the occasional instillation of drops of

℞.	P. Ac. Boric.	grs. 8.
	Spt. Vin. Rect.	ʒiv.
	Aq. ad	℥i.—M.

Otomycosis. Growth of Fungus, or Mycosis of the External Auditory Meatus.—The most common fungus growths of the external meatus are the aspergilli—niger, flavus, or fumigatus.

Their growth is said to be favoured by exposure to dry winds, such as those of Egypt (Wingrave), or by residence in a "damp, mouldy locality" (Politzer) They may exist without causing any disturbance, but when the hyphæ, burrowing through the epidermis, reach the Malpighian layer, they set up irritation and often severe shooting pains with tinnitus and deafness. The growth has been known to bring about perforation of the membrana tympani.

A thin, watery discharge from the canal accompanies the pain and irritation commonly experienced. On examination, the tympanic membrane and deep meatus are seen to be covered with a membrane which has spots upon it like fine coal-dust or lycopodium powder. Underneath the membrane, the skin is red, swollen and excoriated in parts.

The diagnosis, surmised on ordinary examination, is established by microscopic evidence of the filaments, hyphæ, and sporangia of the fungus.

Treatment.—After it has been syringed, the meatus is filled with warm alcohol, which is allowed to remain there for a quarter of an hour. Or

℞.	P. Ac Boric.	grs. 8.
	Spt. Vin. Rect.	ʒiv.
	Aq. ad	℥ i.—M.

may be instilled in drops.

The latter is useful if the patient finds pure alcohol too painful, but the alcohol bath is the more rapid and certain cure.

One or two applications only are required as a rule, although to prevent relapses, the alcohol should be inserted weekly for a time.

External Otitis Secondary to Middle-Ear Suppuration should be considered along with that disease. In such cases, note, on inspecting the canal, whether there appears to be any bulging of the deep postero-superior wall. If so, the case is primarily one of middle-ear suppuration complicated with external otitis and mastoiditis, and the attention is to be fixed on the mastoiditis. If the mastoid is free from disease, the external canal may be protected from irritation by coating its walls with a bland emollient like ungt. ac. boric. before instilling drops for the middle ear.

Should the external otitis produce so much narrowing of the meatus as to interfere with the treatment of the middle-ear disease, a mastoid operation is necessary in order to lay open the middle-ear spaces.

Syphilitic Condylomata of the external auditory meatus occasionally appear during the secondary stage of syphilis in ears which are the seat of chronic discharge, the continual moisture favouring the formation of the excrescences.

The meatal walls are red, thickened, lobulated, and raw on the surface, and the lumen of the meatus is obliterated. Pain is a common feature of the condition.

The *Treatment* is that of syphilis (see p. 71). Locally, calomel should be dusted over the growths.

IMPACTED CERUMEN

in the meatus is perhaps the commonest cause of deafness. Its occurrence is predisposed to by any condition of the middle-ear or meatus which provides a nucleus around which the normal ceruminous secretion can collect. This occurs, for example, in cases of healed suppuration of the middle-ear in which a slight secretion from the ear takes place, drying into crusts or flakes in the meatus. Thus, the writer has found evidence of old suppuration in nearly one-half of the cases of impacted cerumen. It is likewise associated with a very chronic desquamative external otitis. Among other causes, we find narrowings and abnormal tortuosities of the meatus; senile atrophy of the meatal cartilage reducing the external orifice to a slit; and finally, the presence of atmospheric dust like coal-dust, which favours the hardening and accumulation of cerumen in the canal.

In a large proportion of the cases, the mass of cerumen is mingled with hairs, evidently shed from the meatus.

Symptoms.—Impacted cerumen produces sooner or later deafness of the obstructive type, frequently sudden in onset, and generally more severe on one side than on the other. In addition to this, the patient often experiences a sense of fullness or of a foreign body in the affected ear, and sometimes autophony may be complained of. In addition, vertigo; pain in the ear and side of the head, at times of a very severe neuralgic character, may be traced to the presence of a plug of cerumen in the meatus. And a hard cough, and even uncontrollable vomiting from reflex vagus irritation are sometimes experienced.

Diagnosis.—The solid dark-brown mass blocking the external meatus ought, of course, to be unmistakable. But cerumen is not seldom mistaken for cholesteatomatous material in the meatus. Probing the mass in the latter case will show that it is soft, and on withdrawing the probe charged with cholesteatomatous material, the characteristic foul odour will at once become perceptible.

Another point of importance to be noted is that if a small ceruminous mass is seen attached to the postero-superior meatal wall and the adjoining portion of the membrane, then a slow-going suppuration is indicated; for this mass is a crust formed of the dried secretion from the middle ear and coloured brown from admixture with cerumen.

The removal of cerumen may be followed by some hyperæmia of the meatal wall, and at times by the formation of a bulla containing blood on the inferior wall close to the membrana tympani. Do not mistake this bulla for a piece of cerumen accidentally left behind.

Prognosis.—The prognosis is good when the deafness is due to the ceruminous accumulation, but it is wise not to promise complete restoration of hearing before removing the plug, as there may be middle ear or cochlear deafness present.

On the other hand, old people with senile nerve deafness combined with blocking of the external meatus by cerumen, may actually be stone-deaf until the cerumen is removed, and then the hearing may be restored beyond all expectation.

Treatment.—If the mass is soft, it may at once be syringed out, but if it is hard, then it is advisable to soften it by the preliminary instillation of sol. hydrogen peroxid. (5 vols. per cent.) for three or four days before syringing. A small aural spud or curette may be employed to ease the mass away from the meatal wall just before syringing. If after repeated syringing the mass still remains in position, the spud may be used to remove the plug, passing it between the meatal wall and the mass in the postero-superior segment of the meatus, and pressing downwards, forwards and outwards; that is, away from the tympanic membrane. (See Fig. 161.)

Syringing should be done under inspection with the metal ear syringe, and moderate force only should be employed. The nozzle of the syringe is introduced within the orifice of the meatus, and care should be taken that it goes no deeper. The auricle during syringing is pulled gently upwards and backwards so as to straighten the meatus.

Some authorities decry syringing and prefer to extract the cerumen, bit by bit, with spud or curette. The method avoids the slight risk of infecting the middle ear if the membrane happens to be perforated; but on the other hand, it is much more painful for the patient, and it demands a thoroughly practised hand if the extraction is to be effected without injury to the delicate meatal walls.

Recurrence.—People who are subject to cerumen frequently accumulating should insert a few drops of the peroxide solution into the ears regularly once a week.

Foreign Bodies in the Ear.—All sorts of articles find their way into the auditory meatus. The commonest is cotton-wool, plugs of which, inserted "to keep out the cold" may lie forgotten in the meatus for years.

Symptoms.—If the canal is not completely occluded, and if the foreign body is composed of bland and non-absorbent material, no symptoms will appear; but in most cases signs of external otitis, more or less acute, quickly manifest themselves. Absorbent bodies, like peas, etc., by swelling up, give rise to severe pain, and later to serous, and finally purulent, discharge. The diagnosis is quite easy if the examination is made before the canal becomes swollen and blocked with pus, granulations, and débris. After that has taken place, however, only very careful cleansing and inspection will lead to the discovery of the source of the trouble. It frequently happens that the patient is quite unaware of the presence of a foreign body in the meatus.

Extreme care must be exercised in examining these cases not to probe the foreign substance in such a way as to drive it deeper into the canal; for if it gets beyond the isthmus, and still more if it is pushed through the tympanic membrane, then not only is its removal a matter of great difficulty, but the after-results, in the shape of middle-ear suppuration with its sequelæ, are, of course, much more serious than if the foreign body had been left alone.

Indeed, it may be laid down as an axiom that a foreign body in the meatus is generally harmless so long as no attempts are made to remove it by amateurs. Unfortunately, we seldom find that foreign bodies are allowed to remain undisturbed until the expert gets them.

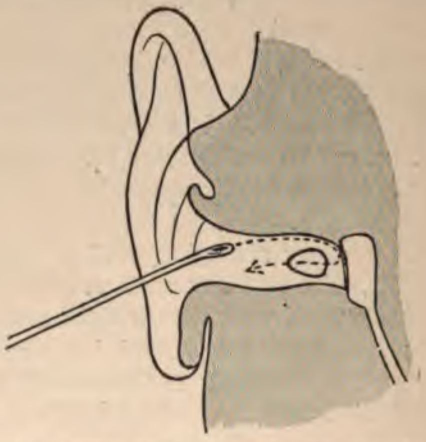

FIG. 161.—Removal of foreign body from the external auditory meatus by ear-spud (diagrammatic—after Laurens).

Treatment.—Most foreign bodies can be removed, like cerumen, by syringing, and in children certainly, no other plan should be tried without a general anæsthetic.

Avoid the use of forceps, particularly for smooth spherical bodies like beads.

If syringing fails to remove it, or if blundering efforts have resulted in septic external otitis, and all that we can see is a mass of granulations exuding pus, then, assuming that the presence of a foreign body is known, operative measures are called for.

Children should be given a general anæsthetic, and the same applies to sensitive adults with an inflamed and tender meatus.

The instrument, and the mode of removal adopted will, to some extent, be dictated by the nature of the foreign body, when that is known. Thus, a small sharp hook or curette passed into the meatus, and dug, from behind, into a soft material such as wood, will sometimes be advisable. Or, again, a blunt hook or curette may be insinuated past the foreign body until it gets beyond it. All manipulations must be cautiously executed.

If these fail, owing to the depth to which the foreign body has been pushed, or owing to its impaction in the meatus, then a post-aural incision must be made, the auricle reflected forward, the membranous meatus turned out of its bony canal, and that canal enlarged, if need be, with gouge and chisel, to such a size as will enable us to remove the foreign body with ease.

It has happened on several occasions that a mastoid operation undertaken for intractable middle ear suppuration has revealed the cause in the shape of an unexpected foreign body, deep in the ear, and concealed by polypoid granulations.

Cutting operations should be avoided if possible, as the mastoid approach and interference with the meatus is liable, especially in childhood, to result in a narrowing of the latter from cicatricial stenosis. In any event, if the foreign body has penetrated the membrane, and has reached the middle ear, the case is naturally serious, and after the removal of the foreign body, for which, in these circumstances, a cutting operation is unavoidable, the surgeon must be prepared to treat the resulting suppuration of the middle ear, with whatever complications may arise.

Insects in the meatus cause a most alarming commotion by impinging upon the membrane. Cockroaches scrape it with their feet; and fleas, in their efforts to escape, leap against it with tumultuous force.

They are easily killed by filling the ear with warm oil, and they can then be syringed out.

Larvæ in the ear sometimes complicate suppuration in neglected children, especially in warm climates. Neither syringing nor the forceps will dislodge them, but oil in which is placed a few drops of " petroleum, turpentine, or ethereal oil " will, according to Politzer, make them crawl out of the meatus.

Osteomata in the meatus may be sessile (*hyperostosis*) or pedunculated (*exostosis*); cancellous or ivory; single, or, more frequently, multiple. They spring either from the osseous meatal wall or from its junction with the cartilaginous meatus. When multiple, they narrow the calibre of the meatus all round; when single, the meatal opening becomes slit-like. Exostoses of the meatus seem to be the result of some long-continued irritation, such as chronic suppuration of the middle ear, or eczema of the meatal wall. And they are very common in people who indulge freely in sea-bathing and diving.

The *Symptoms* are those of meatal obstruction, to which may be added, when the obstruction becomes absolute, the signs of acute inflammation of the middle ear from the retention of secretions behind the obstruction. Not until the meatus is quite closed is hearing seriously interfered with. The outgrowths may be found close up to the tympanic membrane, but apparently they do not affect the intratympanic or labyrinth osseous structures. If, therefore, deafness is present, when the meatus is not entirely occluded by the growths, its cause must be sought for in some change other than the osteomata.

Prognosis.—The rate of growth is always slow, and in many the outgrowths seem to undergo no increase in size for years at a time. In others, however, they gradually lead to an occlusion of the meatus. The frequent accumulation of cerumen behind the exostoses, however, brings the patient to seek relief from the deafness resulting therefrom, and usually leads to the discovery of the bony growths before they have entirely closed the meatus. The ceruminous obstruction requires removal with the curette or spud, since syringing will generally fail to dislodge it.

Diagnosis.—As the bulging meatal wall may be covered with cerumen, these growths may be mistaken for simple ceruminous accumulations; but on clearing this secretion away, an ivory-white or pale-pink, glistening, smooth, hard body is disclosed, filling the meatus more or less, and attached at one side. The surface may be red and inflamed, or even eroded and bleeding, from rubbing with the finger. When multiple and growing from the deeper portions of the canal, we may mistake these bony exostoses for simple bulgings of the membrane. The pale colour and hardness are distinguishing points.

Treatment.—Unless the bony growth has occluded the meatus, it should be ignored, arrangements being made to have the meatus periodically cleared of cerumen and débris, and inspected.

If the meatus is occluded by the growth, it must be removed in one of two methods according as the osteoma possesses a pedicle or is sessile.

If pedunculated, often all that is required is a smart tap with a mallet and chisel, as this breaks the slender stalk across, and enables us to remove the small sphere of bone.

If sessile and multiple, the patient is anæsthetized, and the canal widened by shaving off the redundant bony outgrowths with a fine gouge and chisel. The operation is one of some delicacy, as it is performed through the meatus, and must be accomplished without damaging the membrane. The chief difficulty arises from blood obscuring the field of operation.

It may happen if the growths are deeply situated in a sharply bent meatus, that operation through a post-aural incision, and after reflection of the auricle forward is necessary. In this case, one enlarges the bony meatus, and indeed in a sense reconstitutes it by a free removal of its bony walls, followed, it may be, by grafting.

The results of this operation are not so good as when it is possible to operate through the undisplaced meatus, as stenosis from cicatricial contraction is a common after-result of the more extensive operation.

Wounds and Injuries of the Membrana Tympani.—*Causes.*—Foreign bodies in the meatus, as we have seen, may be pushed through the membrane by incautious attempts at their removal. Or they may be driven through it when they first enter the canal. A blow on the side of the head which induces a sudden rise of air-pressure in the meatus, and violent aërial shocks, such as are produced when heavy artillery is fired close to the ear, may cause perforation. The same is true of shell and bomb explosions, such as those the war has familiarized us with. The displacement of air from a bursting shell affects most seriously the ear which is turned towards it, but naturally the effect depends upon a number of factors, of which distance and exposure are the most important. Thus the damage inflicted upon the ear varies from the total physical destruction to simple propulsion inward of the intact tympanic membrane—that is to say, in so far as the membrane itself is concerned. The effect of war injuries upon the middle and internal ear will be considered later. But we may here remark that the curious observation has been made, that when the shell-burst tears the tympanic membrane, the labyrinth has a better chance of escaping injury, and *vice versâ.*

Lastly, fractures of the middle fossa of the base of the skull implicating the petrous portion of the temporal bone may be accompanied by rupture of the tympanic membrane, and the escape of cerebro-spinal fluid from the ear. The writer once produced a minute rupture at the umbo of the handle of the malleus in an atrophied membrane, while inflating through the Eustachian catheter in the ordinary way with a handbag; but rupture produced in this manner is excessively rare. Finally, ear-pulling, by dragging on the cutaneous lining of the external meatus and through it on the membrana flaccida may, it is said, tear this portion of the membrane.

The direction of traumatic perforation of the membrane runs, as a rule, vertically up and down, and is more or less linear, although if the membrane has been taut, the opening may gape. But this appearance is not invariable. The perforation may be irregular, or radiating in fissures from a point, or even punched out, or the whole membrane may be destroyed.

Symptoms.—Save in the violent, overwhelming experience of a shell-burst, when all minor sensations are blotted out, the patient is made aware of the rupture of the membrane by immediate and severe pain, coupled with a loud subjective noise, and followed by deafness and tinnitus more or less severe. At first, there may be some vertigo, and even faintness and shock.

On inspection, bleeding will be seen if the rupture is recent, but it is not severe save when the rupture is the result of a fracture of the base of the skull, in which case the blood pours out of the ear.

Frequently, and this applies specially to shell cases, the injury to the ear is overlooked by the surgeon, and ignored by the patient until the onset of reactionary otitis draws attention to it.

When the rupture is seen immediately after, or within a day or two of its infliction, one can see a vertical, more or less sharp-edged, though rugged tear, and through it the pale pink colour of the normal tympanic mucosa is visible. This fact enables us to distinguish between recent tears and old perforations from disease, as in the latter the tympanic mucous membrane is red and inflamed.

If neglected or improperly treated, the middle ear becomes the seat of suppuration, and the case passes into the category of suppuration of the middle ear, with all its possibilities. This change is liable to set in about a week or ten days after the injury has been received, and in war cases, it has been found in about 30 per cent. of the cases (Marriage).

If suppuration does not take place, the rupture heals in from one to three months with good hearing. If suppuration does occur, the recovery is, of course, indefinitely postponed.

Diagnosis.—When seen shortly after infliction, the appearances are characteristic, but subsequent appearances depend upon whether suppuration has occurred or not.

The hearing should be carefully tested, and the presence of any degree of nerve deafness regarded as indicating injury to the labyrinth, provided that the patient was not previously deaf.

Prognosis.—If diagnosed and treated early, the prognosis as regards the injury is good; it is good also as regards hearing when there is no nerve deafness. Nevertheless a degree of permanent deafness is not uncommon.

Treatment.—If seen shortly after the accident, disinfect the meatus with biniodide of mercury and spirit (1-1000), mopping it out with the antiseptic under inspection. After thorough cleansing, plug the meatus with sterilized wool, and leave it alone.

Syringing and drops should be avoided.

The same treatment should be adopted for rupture of the tympanic membrane from fracture of the base of the skull, with all the more punctilio, seeing that the fatal septic meningitis of such an accident may be set up by infection from the external auditory meatus.

Strictures of the External Auditory Meatus, and Atresia of the Meatus.

Strictures of the canal are caused by inflammatory swelling of the walls by eczematous infiltration; or they appear as a sequel to suppuration of the middle ear; to external otitis; to ulceration and subsequent healing of the meatus; and to cicatricial contraction from whatever cause arising, as from operation. In old people, the walls of the canal at the orifice tend to come together from atrophy of the meatal cartilage.

Strictures due to inflammatory swellings subside on the removal of the cause. Cicatricial narrowings, on the other hand, are permanent. They may be thin, and present an annular opening, so as to resemble the tympanic membrane with a perforation in it. They are distinguished therefrom, however, by their proximity to the meatal orifice.

Osseous strictures are sometimes seen after long-continued suppuration of the middle ear.

Treatment.—Attempts may be made to dilate soft strictures, with graduated tents or tubes, but when the narrowing of the canal obstructs hearing seriously, or, as is more often the case it impedes the free discharge of pus from the middle ear, then a radical mastoid operation alone is capable of meeting the wants of the case.

Atresia of the External Auditory Meatus is due generally to old middle ear suppuration, perhaps with necrosis and exfoliation of a sequestrum. The condition is produced by the organization of granulation tissue which has entirely filled up the tympanic spaces together with the deep meatus. In many of the cases the connective tissue ultimately becomes converted into bone, and all we can see on examination is a short, blind canal, entirely lined with skin, and feeling at its fundus hard and resistant to the probe.

In these bony cases, the hearing is generally gravely impaired, and the question of treatment turns upon this fact. If the audition, says Politzer, is good enough to let the patient hear a whisper through a hearing-trumpet, then it is worth while trying to remove the atresia by operation. But if the hearing is not as good as this, then the atresia is bony and operation is not likely to improve the audition.

THE TYMPANIC MEMBRANE

The tympanic membrane participates in the diseases both of the external meatus and of the middle ear. In addition to the changes which it manifests in these affections, an acute inflammation, limited to the membrane (acute myringitis) is sometimes seen.

Acute Myringitis begins with congestion of the outer layer of the tympanum, and leads on to an exudation under the skin layer, producing one or more bullæ containing blood-stained or watery serum, or, in the severer cases, pus.

The *symptoms* are shooting pains in the ear, which tend to pass off, as the bullæ form, unless abscess in the substance of the membrane develops. Pyrexia is not present, save perhaps occasionally in children; the hearing is but little affected, and the duration of the attack is brief, the patient being restored to health in three or four days. Middle ear involvement is unusual.

The *diagnosis* raises the question of acute otitis media, as the red, inflamed and bullous condition of the membrane has the same appearance as is presented in some of the most severe types of influenzal otitis media. In middle ear disease, however, the hearing is considerably impaired from the outset, and this point alone is sufficient to render mistakes improbable.

Treatment.—The treatment is the same as that of acute otitis media (q.v.). If bullæ form, and the pain is severe, it may be possible to prick them with a needle, care being taken not to pierce the membrane altogether.

For *Ruptures* and *Perforations* of the membrana tympani, see pp. 459 and 485.

THE MIDDLE EAR

The Eustachian Tube—Eustachian Catarrh—*Causes.*—The ordinary dullness of hearing which so frequently accompanies nasal and naso-pharyngeal catarrhs is due to Eustachian catarrh, the process spreading up from the naso-pharynx. Its occurrence is favoured and its continuance ensured by the presence of adenoids in the naso-pharynx. The deafness and other symptoms are due to a blocking of the Eustachian tube by the catarrhal swelling and secretion, in consequence of which the middle ear is shut off from the pharynx, so that the air in the tympanic cavity is not renewed from time to time after the normal fashion. The air enclosed in this space is then quickly absorbed, and the positive atmospheric pressure on the outer surface of the drum membrane, coupled with the negative pressure within the cavity, results in the indrawing or retraction of the membrane, with crowding-in of the ossicles. Deafness, a feeling of fullness in the ear, and tinnitus ensue; vertigo may also be experienced, but it is uncommon. These interferences with function are, of course, due to the partial immobilization of the ossicular chain, and to the disturbance of the normal relationship of the several members of that chain to one another. Associated with this, we may have some direct interference with the activity of the labyrinth, produced by the increased pressure of the foot-plate of the stapes in the oval window.

Inspection.—The membrana tympani is indrawn, and the light-reflex distorted, but the colour, texture, and lustre of the membrane are, as a rule, unaltered. There is an exception to this statement, however. Sometimes the membrane, stretched and thinned out, may appear more transparent than in health, so that the underlying structures may be visible; but there is never any abnormal redness of the membrane unless the catarrh has extended to the middle ear. Examination with the naso-pharyngoscope often reveals swelling of the orifice of the tube in the pharynx. (Plate II. Fig. 1.)

Functional Examination will reveal the phenomena of obstructive deafness (q.v.). Certain modifications of typical obstructive deafness are, however, not uncommon. In patients over forty years of age the bone conduction, already somewhat reduced, may not appear increased compared with the normal of a younger man; and the labyrinth impairment alluded to above will be manifested when there is a lowering of the upper tone limit, as tested by Galton's whistle. In short, there may be an amount of "nerve deafness" present.

Inflation.—It is no exaggeration to say that the diagnosis of Eustachian obstruction rests chiefly upon what we learn from inflation.

Politzerization is either negative, when the obstruction is considerable, or imperfect, when the obstruction can be partially overcome.

Inflation through the *catheter* produces a low-pitched, harsh note, which may suddenly sound nearer to the listening ear when the obstruction is overcome. Thus, inflation shows that there is obstruction. Not only so, but the inflation, if it has been successful, is attended by an instantaneous removal of the deafness and relief of the other symptoms. Of course this relief is only temporary, but from the point of view of diagnosis, its occurrence is of great value.

In cases of Eustachian obstruction, whether in children or in adults, examine for adenoids.

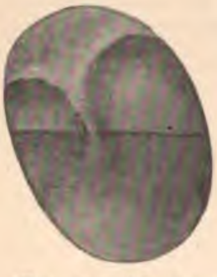

FIG. 162.—Membrana tympani with fluid in the tympanic cavity.

Some deviation from the typical appearances of the membrane just described may be noticed in Eustachian catarrh. Thus it occasionally happens that, as a result of the obstruction in the tube, fluid secretion may be dammed back and accumulate in the tympanic cavity, without any catarrh of the middle ear itself. In these circumstances the presence of fluid in the middle ear will be indicated by the lower segment of the membrane appearing duller in colour than the upper segment, and in addition, a fine traverse line running across the membrane will be visible on careful inspection; this line marks the upper level of the fluid. (Fig. 162.) Auto-inflation, practised by the patient, may, if the air passes the Eustachian obstruction, produce among the fluid tiny circles or bubbles of air, which may be visible through the membrane on examination with the speculum.

The above description applies to what may be termed acute or subacute Eustachian catarrh. In chronic Eustachian catarrh the causes, signs, and symptoms are identical with those of the acute form of the disease, and the difference is only one of duration.

If catarrh in the tube spreads up to involve the tympanic cavity, the appearances presented by the membrane will approximate to those of sero-mucous catarrh of the middle ear, an account of which follows.

Prognosis.—The prognosis of simple Eustachian catarrhal deafness is good, provided the cause can be removed. The disease is typically seen in children and young adults, forming, indeed, one of the signs of adenoids. Consequently it is curable by the removal of these growths. Caution, however, must be exercised in expressing a prognosis not to be too confident that hearing will be entirely restored, as it occasionally happens that deafness persists after operation.

Treatment.—This, and the closely related exudative catarrh of the middle ear are the varieties of deafness which respond most readily to suitable nasal and naso-pharyngeal measures. Adenoids should be removed, and especially those which cluster in the fossa of Rosenmiller behind the naso-pharyngeal orifice of the Eustachian tube. Posterior ends of the inferior turbinal, if enlarged, should be snared. Deflected nasal septa should be straightened by submucous resection.

Thereafter, if the deafness continues, a course of Eustachian catheterism should be undertaken. The catheter is passed once or twice a week, the ears inflated, and the following mixture injected and blown into the middle ear through it.

℞.	Iodin. Resublimat.	grs. ii.
	Menthol.,	grs. iii.
	Ol. Eucalypt.,	♏ iii.
	Ol. Sassafras.	♏ i.
	Paraff. Liq. ad	℥ i.—M.

Argentum colloid, or iodine colloid are also used for the purpose (Mark Hovell).

In children in whom catheterism cannot be accomplished, one must use the Politzer bag, and in cases where the deafness is not cured by the operation for adenoids, it is well to instruct the mother or nurse how to use the bag, as by inflating the ears in this way once or twice a week, more especially at periods when the deafness is severe, it is often possible to obtain a complete cure. In little children, Politzerization can be accomplished when they are crying.

THE SEPTIC INFECTIONS OF THE MIDDLE AND INTERNAL EAR, AND THEIR COMPLICATIONS

Acute Catarrh of the Middle Ear. (Acute Catarrhal Otitis Media.)

Causes.—Acute catarrh of the nose and naso-pharynx from cold, measles, etc., may induce acute catarrh of the middle ear.

In this disease, the brunt of the inflammation is borne by the tympanic cavity rather than by the Eustachian tube, but both may be implicated.

The extension of acute catarrh to the middle ear results in the occurrence of inflammation of the mucous lining and the exudation of a sero-mucous secretion.

The disease is thus due to an infection just as is acute suppuration, and the difference between acute catarrh and acute suppuration is, to begin with, simply one of degree. In catarrh, the infection and reaction are milder and more superficial; in suppuration the infection is more virulent, and its effects more severe.

Symptoms.—Earache, which may be acute, particularly at night, comes on somewhat suddenly, and is accompanied by deafness and tinnitus. In children, all the subjective and constitutional symptoms are more severe than in adults, and pyrexia is a usual accompaniment. After a period of from five to ten days, relief to all the symptoms is afforded by the rupture of the membrane, and the discharge from the middle ear of serous exudation mingled with a little blood. Unless antiseptic precautions are taken, infection of the tympanum through the perforation may take place, and transform the catarrhal into a purulent inflammation. If measures are taken, however, to prevent this complication. the disease runs a short and favourable course—the discharge soon dries up, the perforation heals, and the hearing is restored.

After recovery, attempts should be made to discover, and, if possible, to remove, any predisposing causes of acute middle ear catarrh, such as adenoids, chronic rhinitis, etc.; otherwise the trouble will recur sooner or later.

Functional Examination reveals "obstructive deafness."

Inspection.—The appearance of the membrane varies with the stage of the disease. In the early stages a simple hyperæmia around the handle of the malleus and in the membrana flaccida, is all that can be seen to indicate the presence of catarrh in the deeper structures.

The membrane may be somewhat indrawn, but never to the extent seen in Eustachian obstruction. In the later stages, the gradual accumulation of fluid exudation in the tympanic cavity leads to a bulging of the membrane. By the time bulging is present, the membrane, as a whole, has become uniformly red and inflamed; so that at this period, particularly in children, it may be difficult or impossible to say whether the inflammation is catarrhal or suppurative. In catarrh, however, as a rule, the congestion of the membrane is less deep than in suppuration. The bulging also is different in the two diseases. In catarrh, the distension by the fluid in the tympanic cavity causes a bulging of the membrane as a whole; in suppuration, the bulging particularly affects one portion of the membrane—viz., the postero-superior quadrant.

After rupture, the presence of a serous or sero-mucous discharge in the canal, and the discovery of a recent perforation in the membrane, make the nature of the case quite evident.

The naso-pharyngoscope generally reveals swelling and œdema of the lips of the Eustachian orifice, and adenoids are frequently visible.

Prognosis.—The acute middle-ear catarrh of coryza usually gets well as the general symptoms subside, and this is true even of the cases in which the membrane perforates, and the deafness also is frequently recovered from entirely.

But the disease has a tendency to recur; or to pass into subacute or sero-mucous catarrh; or to become seriously infected and purulent. And these tendencies are most liable to develop when there is any naso-pharyngeal or nasal catarrh or suppuration.

Treatment.—The patient should be put to bed, or at least kept indoors. A mild aperient is given. In children, gr. i. of calomel answers the purpose.

If there is coryzal rhinitis, that must be treated (p. 258).

Earache from acute catarrh is best combated by the instillation into the meatus of the infected ear of

℞.—Glycerin. Ac. Carbol. ʒ ii. ss.
Glycerin ad ℥ i.—M.

warmed before use, and retained with a plug of warm wool in the meatus. The patient lies on the affected ear on a rubber hot-water bottle, a pad of wool being interposed to mitigate and regulate the heat.

At this stage the application of two or three leeches on the mastoid process will sometimes cut short the threatening symptoms and obviate the perforation or incision of the membrane. If this fails, however, and if the membrane is seen to be red and bulging, it should be incised lest the suppuration should develop. But if there is bulging without redness and without pain, the catheterization of the Eustachian tube will probably suffice, in adults at all events, to relieve the symptoms.

After the membrane is opened, the drops of dilute glycerine of carbolic acid are continued until the discharge stops.

If, subsequently, the deafness does not get well, it will be advisable to use the Eustachian catheter as described on p. 432, or, if skilled assistance cannot be obtained, to inflate with Politzer's bag.

When the ear disease has been cured, attention should be directed to the naso-pharynx (adenoids), nose (chronic rhinitis, turbinal hypertrophies, polypi, sinus suppurations, deflected septum), and pharynx (tonsils), so as to prevent a recurrence of the disease.

Suppuration of the Middle Ear.

Suppuration of the middle ear is the most dangerous to life of all the commoner diseases of the ear.

(1) ACUTE SUPPURATION (Acute Suppurative or Purulent Disease or Catarrh of the Middle Ear; Acute Purulent Otitis Media; Otitis Media Purulenta Acuta).

Suppuration of the middle ear is termed "acute" when the case is seen before the membrane has ruptured, or when the discharge has been in existence for a very short space of time—a fortnight to a month.

The distinction between acute and chronic suppuration of the middle ear is largely clinical. But pathologically, the former is recognized as the period of mono-microbic infection.

Causes (*Predisposing*).—Any cause which facilitates the conveyance of extraneous organisms to the Eustachian tube and middle ear, and their retention there, predisposes to suppuration. Thus, in the naso-pharynx, adenoids and naso-pharyngeal tumours ; in the nose, purulent disease ; in the ear, perforations in the membrane, whether old or recent, which lay the feebly resisting tissues of the middle ear open to attack from the organisms normally present in the meatus, are the commonest predisposing causes of aural suppuration.

The disease is favoured by any condition, local or general, which lowers the resisting power of the tissues. Thus, it is a frequent accompaniment of the acute catarrhal fevers or " colds " ; of scarlet fever, influenza, measles and other infectious fevers, and also at times of pneumonia.

Infection of the middle ear leading to suppuration also follows a traumatic rupture of the membrane the treatment of which has been neglected. It may be initiated by foreign bodies, and by the discharge of a furunculosis breaking through the membrane.

From the naso-pharynx, acute suppuration of the middle ear may follow the improper use of nasal douches, the fluid, and with it, perhaps, some purulent nasal secretion, washing up the Eustachian tube, and infecting the middle ear. Sea-bathing is also an occasional cause of the disease, the sea-water, which at most bathing resorts is charged with septic organisms, obtaining an entrance to the middle ear, either through the nose and naso-pharynx, or it may be through a perforation in the tympanic membrane.

Finally, the disease may occur as a sequel to operations on the naso-pharynx.

The *organisms* most frequently responsible for acute suppuration are the streptococci, and the pneumococcus, and before or immediately after the membrane ruptures or is incised culture will nearly always show the infection to be pure. In addition to these, however, the micrococcus catarrhalis, the diphtheria bacillus, or the influenza bacillus may prove to be the causative organism. And of late years, attention has been drawn to what is called the *streptococcus mucosus*, a capsulated organism, which has been found to be responsible for a peculiarly obstinate and virulent form of the disease, often resisting all attempts by operation to eradicate it, and marching relentlessly on, to terminate in meningitis. Fortunately, this particular infection is not common, but when it is found, the prognosis must always be regarded with apprehension.

Staphylococci also are said to occur in pure culture sometimes in acute suppuration of the middle ear, but more frequently they are evidence of a change in the nature of the infection, being found some time after the membrane is opened.

Regarding the relationship of acute catarrh to acute suppuration, it is generally supposed that in order to produce tissue necrosis and suppuration, either the organism must be specially virulent, or the tissues of specially feeble resisting-power, or there is a combination of both circumstances. If the microbe is feeble, catarrh will result, and not suppuration. In catarrhal inflammation, the disease is altogether milder, and is limited to the superficial layers of the mucous lining of the tympanum, while in suppuration, the reaction is, as a whole, more intense, the inflammation involves the mucous, submucous and periosteal layers, and not infrequently the bone itself, while extensions and complications are usual.

Pathology.—In most cases the infection reaches the middle ear spaces from the throat, travelling up the Eustachian tube in the tissues, as well as on the surface of the mucosa, and inducing in its passage an inflammatory swelling of the tube, which seals it, and closes off the middle ear spaces from the naso-pharynx, thus blocking the natural drainage outlet of the middle ear. In the antro-tympanic spaces, the site and extent of the inflammatory process varies with the virulence of the infection; with the local and general resistance, and with the anatomical conformation of the middle ear, and of the mastoid process.

But in all cases the chief centre of the disease is at first in the folds of mucous membrane around the chain of ossicles in the attic, and in some cases the focus remains limited to this region.

In most cases, however, the disease involves the whole of the tympanic cavity, producing inflammatory œdema, and an exudation of the muco-pus, with shedding of the surface epithelium into the cavity of the middle ear, the accumulation of which distends the tympanic membrane, causing it to bulge towards the external auditory meatus, and eventually to rupture into that canal, with relief to the symptoms through providing a drainage that, in the majority of cases, leads in a few days or weeks to natural cure.

This fortunate termination, however, is by no means to be relied upon, as the variations, complications, delays and dangers of the disease are manifold and frequent.

Variations.—At one extreme we meet with cases in which the membrane, after a few days of inflammation, ruptures, discharging some blood-stained serum, and then promptly heals up again for good. At the other extreme stand these cases, commonest in influenza and scarlet fever, in which considerable areas of bone undergo necrosis, with all the tedious and dangerous sequelæ of that event. And between those extremes, all kinds of variations in severity are met with.

Complications.—The complications of middle ear suppuration consists in an extension of the infection to the labyrinth (labyrinthitis) ; to the mastoid cells (mastoiditis and mastoid abscess) ; to the soft tissues of the neck (" descending " abscess) ; to the lateral sinus (sinus thrombosis) ; to the cerebellum (cerebellar abscess) ; to the cerebrum (temporo-sphenoidal abscess) and to the meninges (meningitis).

Among the less common complications are facial paralysis ; paralysis of the sixth cranial nerve ; pterygo-maxillary and pharyngeal abscess ; cranial osteomyelitis ; and pyæmia. Pyæmia is an occasional accompaniment of lateral sinus thrombosis, but as a direct sequel to middle ear infection it is rare.

Symptoms.—The early symptoms before perforation takes place and the discharge appears are the sudden onset of pain, generally severe, with marked pyrexia (temperature 101° to 103° F.), headache and general malaise. Deafness, tinnitus, and vertigo are experienced. In adults, however, acute inflammation of the middle ear, even when leading on to suppuration, may be almost or quite painless, with constitutional symptoms correspondingly slight. Deafness and tinnitus are in these cases the symptoms for which relief is sought. In children, on the other hand, the symptoms, both local and general, are as a rule severe. The pain may be intense. There is high fever, with vomiting, nocturnal delirium, and even strabismus, so that a suspicion of meningitis may arise. **In young children, who cannot express what they feel, it should be a rule in all pyrexial attacks to make a careful examination of the ears with speculum and head-light, just as we always examine the throat.**

In most cases, after a period varying from twenty-four hours to three or four days, the pain and fever are relieved by the bursting of the abscess through some part of the membrana tympani into the meatus. The relief to the pain and other symptoms is, however, less marked than in acute catarrh. After this has happened there is, of course, an end to doubt as to diagnosis. The discharge at first consists of a scanty blood-stained fluid, later becoming more plentiful, but still thin and watery until after the lapse of one or two days, when it becomes definitely purulent.

On examination the appearance of the membrane varies according to the stage reached by the disease. At the outset, all we see is a marked redness with, perhaps, some fullness affecting the membrane flaccida and the region of the malleus handle, while the membrane, in its lower hemisphere, presents the grey colour of health.

At a later stage, when the disease is more advanced, the redness involves the whole of the membrane, which also shows more pronounced œdematous fullness, while the general swelling and bulging of the membrane, wholly or in part, leads to a complete disappearance of the normal landmarks. The anterior and posterior folds, the handle, and even the short process of the malleus can no longer be distinguished, and, as by this time the meatal wall is also more or less involved in the inflammation, it is difficult, apart from position, to determine what is membrane and what meatal wall.

When the process is severe, as it usually is in children, there occurs at this period a desquamation of the epidermal layer of the membrane, and as it lies, a whitish pellicle on the membrane, hasty examination, or examination with insufficient illumination, may mislead us into thinking that the membrane is of a normal colour.

In order to see the membrane in such cases, this sodden epidermal layer must be carefully removed with an ear-probe protected with cotton-wool. And as this proceeding is one of great delicacy, it can only be carried out in children under a general anæsthetic.

The bulging affects the membrane as a whole, but it often happens that it is most pronounced in the upper zone, corresponding with the chief focus of the inflammation. The commonest spot for spontaneous perforation to occur is in the anterior half of the membrane, but perforations of the posterior half are common enough. It is said that the pus is led to the latter quarter by the long process of the incus.

The actual position and size of the perforation can only be made out after the subsidence of the inflammation. Immediately or shortly after perforation, all we can see is a red membrane devoid of all landmarks, forming an acute angle with the anterior meatal wall, and in the depths of this angle a little blood-stained mucus or sero-pus is evident, pulsating as we watch it. On wiping this away with the probe, we can see it slowly re-accumulating.

In addition to the cases in which the state of the membrane as we have just described it, testifies to a general tympanic inflammation, we encounter others in which what looks like a hernia of the membrane is present in the shape of a nipple or sausage-like protrusion of part of the postero-superior segment.

Functional examination during an attack of acute suppuration of the middle ear reveals obstructive deafness, slight in the initial stages, but becoming more pronounced as the exudation forms and accumulates.

The existence of well-marked nerve-deafness should be regarded as a danger-signal, indicating extension to the labyrinth (see later), but a slight falling off in the bone conduction may be ignored.

Course and Terminations.—The majority of attacks of acute suppuration get well spontaneously after the membrane ruptures and the exudation is discharged. The usual history is that immediately after rupture the patient experiences a sense of relieved tension in the ear, this is followed by a subsidence in the intensity of the pain, and by a fall in the temperature to the normal if it has been raised. Pain and pyrexia generally disappear within a few days after the membrane ruptures. The discharge, at first blood-stained and watery, becomes thicker and purulent ; then after a few days it becomes thinner again, and finally dries up entirely. During this period, the deafness, noticeably less severe after rupture, gradually passes off, and the hearing is completely restored. Examination of the membrane during the period of recovery will show the swelling and redness to be gradually diminishing, while the membrane seems to retreat, and to assume more of its normal position, and the landmarks once more become visible. The perforation may now, for the first time, come into sight, but it also participates in the healing process, and at the end of ten days or a fortnight will have cicatrized over entirely, its place being taken by a red, congested area on the membrane, which otherwise is now resuming its normal pearly gray colour.

This fortunate termination of acute middle ear suppuration attacking a previously healthy ear is the commonest event, and it is the termination most likely to be reached when the case is properly treated. But such rapid and complete recovery cannot always be secured, even when the disease is carefully treated, and, if it is neglected, the patient's chances are always less promising. Either the disease drifts into chronic suppuration, or one of the graver complications sets in.

While the transition from acute to chronic suppuration is favoured by neglect or by improper treatment, it is, on the other hand, inevitable where the initial infection has led to osseous necrosis, of one of the ossicles, for example, or of part of the bony walls of the antro-tympanic cavities.

Apart from chronic suppuration, acute purulent otitis media may terminate in one of the complications of septic ear infections, a list of which we have already given at p. 471, and which are described below.

Diagnosis.—Before rupture of the membrane, suppuration is distinguished from acute catarrh by the greater severity of the constitutional symptoms, by the greater pain, and by the deeper redness and greater swelling and bulging of the membrane.

It is distinguished from acute myringitis by the presence of deafness.

After rupture of the membrane and discharge of the secretion, the presence of pus, at all events after the first twenty-four hours, makes the nature of the case clear. But it is important to make sure that the discharge is actually purulent. In herpes zoster oticus, for example, there is swelling of the membrane and canal, together with severe pain and discharge, and perhaps with facial paralysis, and the observer may mistake it for acute suppuration. The distinction lies in the discharge being serous in herpes, while the herpetic vesicles, if seen, would prevent confusion arising.

Furunculosis of the meatus resembles acute middle ear suppuration in being painful and in causing purulent discharge from the meatus, while the mastoid region may, in furunculosis, become swollen and œdematous, and so give rise to suspicion of mastoid abscess. But in furunculosis, the swelling and inflammation are found on the meatal wall external to the isthmus, while in middle ear suppuration, if the meatal wall is swollen and inflamed, the swelling involves the roof and postero-superior wall of the canal, and it can be traced in towards the membrane, into which it is seen to merge. In furunculosis, on the other hand, the meatal swelling, intense in the walls of the outer meatus, ceases abruptly at the isthmus, and examination of the cleansed meatus through a fine speculum, pushed beyond the meatal swelling, will show the membrane to be normal, or but a little reddened, and with all its landmarks visible, while, at the same time, the deep meatal walls are smooth and free from fullness.

One very important point in the diagnosis of genuine middle ear suppuration must not be forgotten, namely, to make sure that the case is actually one of acute suppuration, and *not an acute exacerbation of a chronic suppuration.* This point may generally be established by close cross-questioning of the patient or his friends, or by examination of the membrane. But it does happen, nevertheless, especially in cholesteatoma, that the patient may have been quite unaware of any previously existing chronic disease.

Prognosis.—Acute suppuration of the middle ear arising in the course of scarlet fever, influenza and diphtheria is more serious than when it is due to an ordinary catarrhal infection. But even in the last variety, danger exists. The evil prognosis attaching to the presence of the streptococcus mucosus has already been mentioned.

The presence in children of such acute symptoms as high fever (104° F.), rapid pulse, delirium, and even in infants of head retraction and squint *before the membrane is opened*, need not be regarded too gravely. But the persistence of such symptoms after the abscess has begun to discharge is of serious import.

The clinical rule is: After the membrane has been opened, whether by Nature or by the surgeon, the pyrexia should fall to normal in from twenty-four to forty-eight hours. Pain may continue for three or four days, if it is not getting more severe; and tenderness, on pressure over the mastoid, may also last for the same length of time without necessarily meaning trouble. But œdema over the mastoid; a temperature running at or above 99°; paroxysmal severe pain; persisting headache; vertigo; cessation of discharge with the other symptoms continuing, *any one* of these renders prognosis grave, inasmuch as they all indicate extension of the disease, to the mastoid cells, or elsewhere beyond the limits of the middle ear spaces. (See p. 493.)

The acute symptoms subsiding, the discharge should dry up in from two to four or six weeks. If it continues after that period, the disease should be looked upon as chronic suppuration, and treated accordingly.

Treatment.—In the initial stages before the discharge has appeared, the patient should receive a dose of calomel, followed in adults by a saline purgative. Locally, we seek to relieve the pain by instilling into the external meatus, by means of the author's spoon (Fig. 170) a few drops of

R.	Glycerin. Ac. Carbolic	ʒii. ss.
	Glycerin. ad	℥i.—M.

The spoon is heated by plunging it for a moment into boiling water, and the drops poured into it are thus warmed before being inserted into the meatus. The patient is kept lying on the sound side, and a plug of warm cotton-wool is lightly inserted into the meatus. At the same time, hot fomentations are applied to the region of the ear and side of the head. Or two or three leeches may be applied to the mastoid process.

If this fails to give relief, opium or morphia internally may be given. But the membrane should be inspected frequently, and if the symptoms remain unabated, and the redness and swelling of the membrane show no sign of lessening, then the membrane should be incised (paracentesis or myringotomy). For this, the myringotome, a long, fine lancet with a bent handle, is advisable, but the operation can be performed with a tenotomy or an iridectomy knife, or even, at a push, with a long, straight Hagedorn needle, held in a pair of pressure forceps, or in a needle-holder.

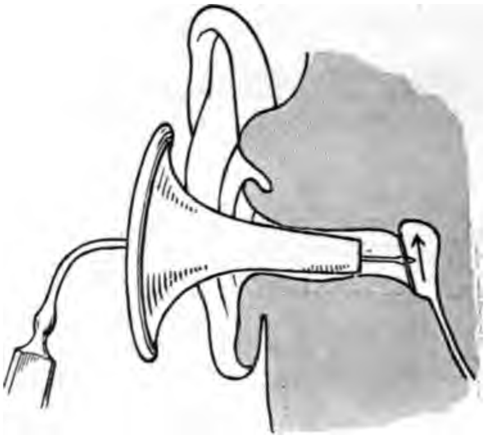

FIG. 163.—Paracentesis Tympani or Myringotomy (diagrammatic)—after Laurens.

Myringotomy (Paracentesis Tympani).—The patient is anæsthetized. Nitrous oxide suffices for adults, but children need chloroform.

The meatus is, as far as possible, sterilized with biniodide and spirit solution. Under inspection with a speculum and good illumination, the red, bulging membrane is identified, and then the point of the lancet is introduced through the speculum to the lowest visible part of the most prominent area of the membrane, and plunged through it into the tympanic cavity. The lancet is carried upwards so as to incise the membrane from below upwards as far as the upper margin of the membrane before it is withdrawn. (Figs. 163 and 164.)

The operation is easy, and practically speaking, free from danger, and its results, on the whole, are excellent.

Pus is seldom seen at the moment of incision, beyond an occasional streak on the surface of the blood that immediately fills the meatus.

This blood is now cleaned out, the carbolic-glycerine drops again inserted, and a fine drain of gauze is passed by the meatus through the incision in the membrane into the middle ear. Boric acid fomentations are applied and changed frequently. The patient is kept lying on the diseased side, so as to encourage drainage.

The gauze drain is removed next day, and need not be re-inserted.

If the operation is successful, the symptoms undergo the subsidence we have already described. The antiseptic drops should be continued as long as there is any discharge.

Healing of the incision is rapid and complete, and as the inflammation diminishes, the hearing returns.

The restoration of function may be hastened by the judicious use of Politzer's bag, the Eustachian catheter, and oto-massage. But in some cases a certain amount of deafness will remain, in spite of all efforts to remedy it.

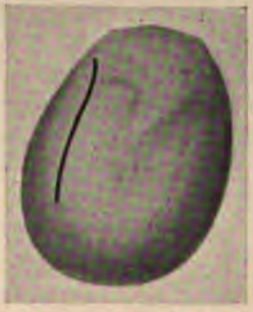

FIG. 164.—Usual site of the incision in myringotomy.

If, after paracentesis, the acute symptoms of fever, or pain, persist; still more if œdema appears, or there is persistent tenderness on pressure over the mastoid process, then the mastoid cells and the antrum should be opened up by the performance of the cortical mastoid operation. (See p. 498.)

(2) CHRONIC SUPPURATION OF THE MIDDLE EAR

Chronic suppuration of the middle ear sometimes supervenes upon acute suppuration; sometimes, as in cholesteatoma (p. 480) and in tuberculosis (p. 517), it is chronic from the outset.

Chronic suppuration will develop from an acute infection of the middle ear spaces when there has been osseous necrosis or caries of the ossicles or walls of the antro-tympanic spaces. Of the ossicles, the incus, by reason of its isolated position, is the most liable to undergo necrosis, but both incus and malleus are liable to caries. The most vulnerable parts of the bony walls are the outer wall of the attic and the meatal wall of the antrum; the promontory; and the bone of the sinus tympani, a small cavity in the postero-lateral region of the tympanum.

Bone destruction of the relatively massive type is most frequently encountered in the acute middle ear infections of scarlet fever, influenza, and diphtheria, and it is in those diseases also that we come across the rarer and more serious and extensive varieties of bony necrosis in which the labyrinth is attacked and more or less of the bony skeleton of the cochlea and semicircular canals undergoes necrosis. In the event of bony necrosis, the disease may rapidly progress to a fatal issue from some intracranial complication, but such disasters are not common, as the protective mechanism is usually sufficient, first of all to shut off the diseased areas, and secondly to lead to an exfoliation of the necrosed bone. This process naturally takes time, and as it is associated with a purulent discharge from the meatus, it is one of the varieties of chronic suppuration.

Apart from such massive lesions as bony necrosis produced during the initial, or at all events, the early days of an acute infection, chronic suppuration may also result from neglect or carelessness in the treatment of a simple acute suppuration, whereby the mono-microbic infection of the acute stage is converted into a polymicrobic infection. This is commonest in children, and especially among children of the poorer classes, in whom eczema of the meatus and auricle is common.

The transition from acute into chronic suppuration is probable, as Arthur Cheatle has pointed out, when the temporal bone with its mastoid process happens to be of the "infantile" or non-pneumatic type, or when the mastoid cells are separated from the surface of the bone by a dense outer osseous cortex. When the tympanic cavities in such a temporal bone become infected, the dense bone of the mastoid tends to limit the disease to the middle ear spaces, and thus the acute infection, if it does not get well, becomes chronic, whereas when the mastoid and other parts of the temporal bone are pneumatic or spongy with air cells, then an acute infection is apt to extend from the middle ear into those cells, setting up acute mastoiditis with abscess formation in and around the mastoid process. The infection may also pass into the petrous and other deeper regions of the bone, but in this type of temporal bone, the tendency is to the production of acute complications, not to a transition into chronic suppuration.

It is necessary to add that the type of mastoid does not invariably determine the development of infective disease. Occasionally one finds acute symptoms with signs of osteomyelitis in a solid or infantile mastoid on the one hand; and, on the other, chronic disease and chronic suppuration may exist in a mastoid which is wholly cellular, from the antrum to the outer cortex and to the tip. But as a rule, Cheatle's contentions will be found to be correct.

Clinically, the moment when a purulent discharge from the ear ceases to be " acute " and becomes " chronic," no one can fix; so that in point of fact, the distinction is rough, save in the case of tuberculous disease, which is chronic from the beginning As a rule, however, it may be stated that when a discharge has lasted longer than a month or six weeks, the case is likely to become one of chronic suppuration.

Pathologically, all suppuration of the middle ear spaces begins with an infection of the mucous lining of the cavities, and in these early stages, the disease, save when the bone is extensively attacked in the varieties we have just been considering, is confined to soft parts. As time passes, the disease extends deeper as well as wider until, it may be, the infection passes into the surface of the bone, and involves the entire superficial area of the antro-tympanic cavity, including the bony segment of the Eustachian tube. In this condition, the middle ear is converted into a series of cavities lined with pus-secreting granulations, and these granulations becoming œdematous and covered with epithelium, form the aural " polypi," so commonly found in chronic suppuration of the ear. With certain rare exceptions, polypus in the ear is, therefore, merely an œdematous granulation, and should be treated as such. That is to say, its simple removal with the snare is an insufficient method of treatment. The cause, in the shape of the suppuration, must also be combated. Such polypi generally spring from the region of the attic, but they may be attached to an exposed area of dura mater, or even to the facial nerve.

When an acute suppuration of the middle ear becomes chronic, the perforation in the tympanic membrane produced during the acute stage of the disease persists, and tends, as time goes on, and the disease continues, to become larger by a process of ulceration, until, it may be, the whole of the membrana vibrans, with the exception of the annulus tendinosus, is destroyed. Shrapnell's membrane, however, together with that part of the vibrating membrane which covers the handle of the malleus, being in contact with bone, and sharing in its blood-supply, generally remains intact, unless, of course, the ossicles themselves are eroded or lost in the course of the disease, in which case, the membrane in contact with them is also destroyed. And it is in consequence of caries of the ossicles that the so-called *attic perforation*, or perforation through Shrapnell's membrane, occurs. (See Fig. 166.)—"Perforation" is perhaps not quite a correct appellation, as this opening is, properly speaking, merely the mouth of a minute sinus leading through Shrapnell's membrane to the seat of the disease in the bone.

Considerable variation in the size and in the rate of enlargement of the perforation in the membrane is presented in different cases; sometimes the destructive process is so rapid that the whole of the vibrating membrane is destroyed in a few weeks; at other times, in spite of the obvious presence of active disease, the perforation remains unaltered in size and shape for months or even years. (See also later, p. 485.)

Cholesteatoma.—What is generally looked upon as a sequel of chronic suppuration of the middle ear is the condition known as cholesteatomatous disease. Cholesteatoma is a foul-smelling cheesy substance which collects in the attic or in the antrum, or, more rarely, in the mastoid cells, and is often found, in neglected ears, filling up the deeper parts of the external meatus. It consists really of a decomposing accumulation of epidermal cells in the ear cavities, and considerable discussion has raged around the question of the presence of epidermal cells in those spaces, seeing that they are normally lined with cubical, ciliated epithelium.

According to Politzer, what happens is, that if there is a perforation of the membrane, and especially of Shrapnell's membrane close to its margin, there is a tendency for the epidermal lining of the external meatus to extend through the perforation, and so to grow and extend over the granulating surfaces that the original cubical epithelial cells of the middle ear spaces are replaced by epidermal cells, which, following their natural process of growth and decay, are constantly being shed or renewed. Inasmuch, however, as they contain horny material, their disintegration and removal cannot take place with the same celerity and completeness that characterizes the physiological disintegration of the normal cells of the cavities which do not contain keratin, and the débris of their decomposition accumulates and forms masses of cholesteatoma.

These epidermal cells and débris are said to find their way into the Haversian canals of the bone, and, whether by simple mechanical pressure atrophy, or by the direct solvent or eroding action of the living epidermal cells themselves, they give rise to absorption or atrophy of the bone, and thus there is produced what clinical experience familiarizes us with, the destruction, namely, by cholesteatoma, of the bony barriers which lie between the middle ear spaces and the adjoining organs and structures.

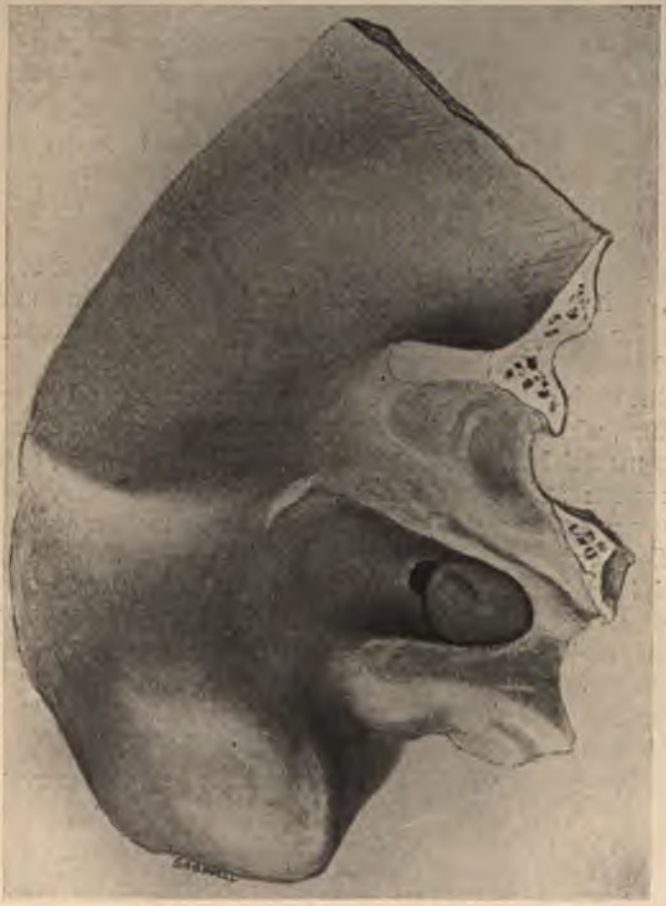

FIG. 165.—Right Temporal bone of adult. The anterior wall of the bony meatus has been removed exposing the tympanic membrane together with a cholesteatomatous fistula of the meatus.

Contrast this aspect of the membrane with that visible through the speculum. Note the malleus handle pointing downwards and *forwards*.

The fistula leads into the aditus ad antrum. Note the relationship of these parts to the supra-meatal spine.

There is no rule governing the direction which this cholesteatomatous erosion takes, but perhaps the most frequent breach in the bone is that of the upper and posterior segment of the deep, bony meatus close to, and sometimes involving, the tympanic membrane. (Fig. 165.) This portion of the meatus is, in reality, the bony partition between the mastoid antrum and the meatus, so that when it is eroded and broken through, the seat of the disease in the cavity of the antrum opens directly into the external auditory meatus, in such a way that a bent probe can be passed, without entering the tympanum, from the meatus into the antrum through the fistulous opening in the bone. By an enlargement of this fistulous opening, which is, in effect, an extension along the meatus externally of the aditus ad antrum, sufficient room may be provided whereby the whole of a cholesteatomatous collection can be spontaneously voided into the meatus, and the disease may thus end in natural cure, with a large dry cavity opening into the meatus, and lined with the epidermal lining which was primarily responsible for the disease.

But such a termination is not by any means to be expected. Because, while the cholesteatoma is producing its erosion of the antro-meatal bony wall, it often is, at the same time, extending by erosion into other and more dangerous regions. Next in frequency to the meatal fistula, comes the formation of a fistula in the floor or inner wall of the aditus into the external semicircular canal, in such a way as to expose the labyrinth spaces to infection, unless, as is usually the case fortunately, there is erected a barrier within the labyrinth against the general infection of its cavities. In like manner, the facial nerve may be exposed in its canal; as also may be the lateral sinus, the dura of the cerebellum, and of the temporo-sphenoidal lobe, so that infection may thus be transmitted to any, or all of these structures, with grave risk to life.

Cases have been recorded, we must add, in which the cholesteatomatous disease of the antro-tympanic spaces seems to have originated without any preceding suppuration of the middle ear, and without any perforation of the membrane, from which it is surmised that the epithelial lining of the normal middle ear may, without preceding suppuration, become transformed into epidermal cells by some cause we are unacquainted with. Cholesteatomatous disease is generally insidious, and may exist without producing enough discharge to attract the patient's attention, and without inducing any considerable loss of hearing.

Symptomatology of Chronic Suppuration of the Middle Ear.—The symptom for which the patient seeks relief is most frequently loss of hearing. Discharge is usually disregarded, and its presence is only revealed in reply to direct questioning or on physical examination.

The *loss of hearing* manifests extreme variability. Sometimes, as in the latent cholesteatoma just alluded to, the loss is so slight as to escape the patient's notice altogether, and his attention is attracted to the ear by an offensive odour, by slight discharge, or, it may be, by the onset of one of the graver complications. In other cases, the loss of hearing is very great.

Examination in simple purulent otitis limited to the middle ear, reveals the usual signs of obstructive deafness. As a rule, conversation and the whisper are heard better than the watch. In old-standing cases, there may be some lowering of the upper tone-limit as estimated by Galton's whistle or by the monochord. Should, however, the examination disclose the presence of decided nerve deafness, the possibility of extension of the purulent disease to the labyrinth must be considered. (See p. 522)

The *discharge* in chronic suppuration is also variable. In a few cases, especially in children, it is so copious as to render the keeping clean of the ear, and even of the neck and cheek, an impossible task. In most cases, however, the quantity is moderate, seldom running out, and easily restrained by occasional cleansing. The discharge often varies in quantity from one time to another, becoming copious when the patient catches a " cold," and scanty in hot, dry weather, when the patient is otherwise well. If periodicity in discharge should, during the time when the ear is dry, be associated with pain or with headache, and if the period of freedom from discharge is terminated by a sudden and considerable flow of pus with relief to pain, or headache, the probability is that we have to deal with an extradural abscess alternately filling and emptying.

If as a result of treatment the disease undergoes improvement, the quantity of discharge will diminish gradually, and ultimately entirely cease. A sudden cessation of the discharge, however, should be looked upon with suspicion, as it may be due to impairment in the drainage of the ear, and foreshadow serious complications.

Cases are frequently found where the discharge entirely disappears for weeks or even months on end. Sometimes these are old cases, in which the disease is gradually getting well, but at other times this behaviour persists unchanged for years. In the intervals, the patient is quite well save, perhaps, for a slight amount of deafness.

The discharge from the ear in chronic suppuration in appearance is muco-purulent, with more or less mucus or pus according to the case. A high mucous content renders the discharge very sticky, and experience associates that feature with very obstinate types of the disease.

Occasionally it is stained with blood from granulations.

Fœtor of the discharge is not unusual, especially when cholesteatoma is present, in which case it is a very prominent feature.

Microscopically, the purulent discharge of middle ear suppuration is seen to contain pus cells with a great number and variety of bacteria of any or all of the pyogenic varieties. It is in this polymicrobic content that the discharge differs bacteriologically from that of acute suppuration, in which, as we have seen, the infection is mono-microbic.

Sometimes, particularly in infected cholesteatoma, as Wingrave has pointed out, the discharge that comes from the meatus contains so few pus cells that it can scarcely be termed pus. It is then composed of serum, squamous cells from the cholesteatomatous cavity, bacteria, and a few leukocytes.

When caries is present, myelocytes may be discovered in the discharge.

Pain.—Uncomplicated chronic suppuration of the middle ear, it may be stated, is a painless disease. Consequently, the presence of pain or earache should be regarded as a signal of some complication. *Vertigo.*—The same remark applies to vertigo, and we shall discuss those symptoms in detail later.

On examination, in order to inspect the membrane and tympanic regions, it is usually necessary to cleanse the meatus of pus, crusts and débris, a little task which is more difficult and delicate than it sounds. The difficulty arises from the adhesive nature of the contents of the canal, the delicacy from the extreme sensibility of the bony meatus, and of the membrana tympani. It is a fact not generally known or appreciated that, as J. G. Wilson has pointed out, the nerve-fibrils on the membrane, like those of the cornea, are naked axis-cylinders. The membrane, therefore, must approach to the cornea in delicacy of sensibility.

The cleansing is best effected by fine cotton-wool swabs on an ear probe, inserted through the speculum under inspection. The wool may be soaked in sol. hydrogen peroxide, but it is often more effective when dry. Syringing is not advisable, as it runs the risk of driving the meatal contents back into the middle ear and adjoining cavities.

The state of matters disclosed after the obstruction has been removed is subject to great variation. The points one keeps in mind are (1) the state of the meatal lumen and walls as regards size and colour; (2) the appearance of the tympanic membrane with regard to colour; to signs of inflammation; and to perforations, with their situation and size; (3) the condition of the tympanic cavity, if that is visible through a perforation; (4) in general, the source and quantity of the discharge; whether granulations or polypi are present; the existence of swollen or bulging regions, with the presence, it may be, of pulsation; (5) the presence of an attic or antrum fistula with or without signs of cholesteatoma.

The Meatus.—The irritation produced by the constant presence of pus in the meatus at times sets up chronic or acute external otitis with narrowing of the canal, or eczema of the meatus and auricle. When the discharge is very scanty, it dries into crusts in the canal, and these crusts may form the nucleus for the accumulation of masses of cerumen.

Any appearance of swelling or bulging of the walls of the meatus, more especially of the roof or of the postero-superior wall close up to the membrane, should be looked for as a routine inasmuch as these appearances mean the extension of the disease beyond the tympanic cavity, or towards the exterior along the meatal roof.

The *Membrana Tympani* may present the colour of health, and except for the perforation, appear to be quite normal. One looks upon this appearance as indicating a mild or subsiding inflammation.

On the other hand, the membrane, when cleaned of its investing secretions and crusts, may be seen to be red, inflamed, and swollen, an appearance most frequently associated with an acute exacerbation of the chronic disease. Under those circumstances, careful search should be made, with a Siegle speculum if necessary, for pulsating areas or spots in the membrane or tympanum, as this indication of tension is frequently a forerunner or an immediate sign of some complication.

Perforation.—As far as we know in all cases of chronic suppuration of the middle ear, the pus reaches the exterior through a fistulous opening, either through the bony walls of the antro-tympanic cavities or through the membrane. In the former case, we have the sinus of cholesteatoma, as we have already seen, while in the latter case, we have what is termed a perforated membrane.

The extent of the destruction of the membrane varies. In some cases the perforation is as small as a pinhole, in others the whole of the vibrating membrane is absent. Large perforations are, of course, easily made out, unless the parts are concealed by granulations or polypi; but it is often a matter of great difficulty to discover the exact situation of a small perforation. In the case of small perforations, if these are multiple and there is an absence of pain, the suspicion will be raised that the disease is tuberculous.

Perforations may be classified as follows :—

1. Small circular perforations, generally behind or in front of and about the level of the umbo of the malleus handle. (Fig. 166.)

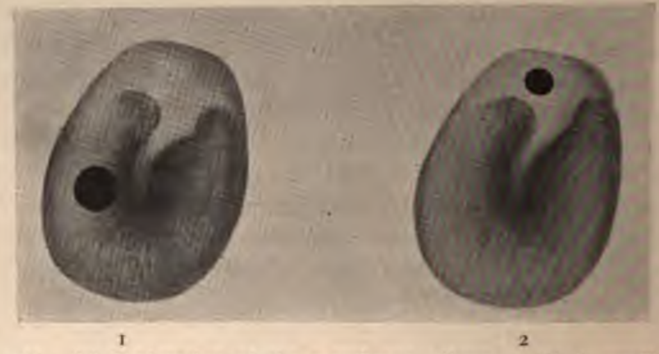

FIG. 166.—(1) Perforation of the membrana vibrans. (2) A small attic perforation.

2. Extensive destruction of the vibrating membrane, the cartilaginous ring alone escaping;—the handle of the malleus is generally tilted horizontally inwards and adherent to the inner wall; Shrapnell's membrane is intact and folded tightly round the ossicles; the *reniform* and *heart-shaped* perforations. (Figs. 167 and 168.)
3. Small perforations through Shrapnell's membrane, usually just above the short process of the malleus with or without granulations. This indicates disease in the bones of the attic, usually in the ossicles, and is frequently cholesteatomatous. (Fig. 166.)
4. Perforation, or fistula, in the postero-superior quadrant of the membrane generally involving more or less of the adjacent bony wall of the meatus, produced by cholesteatomatous disease of the antrum. (See Figs. 165 and 169.)

This last variety is apt to escape detection, as it is situated very obliquely towards the examining eye, and is often associated with a membrane, which otherwise is entire, and is normal or nearly normal in colour. The presence of such a perforation may be looked for when the discharge is highly fœtid, and it is easy to demonstrate its existence by passing into it from the meatus the point of a fine probe bent at right angles. This manipulation requires care, as the parts are exceedingly tender. Siegle's speculum may also reveal it, and may be made to aspirate from it the ice-cream-looking cholesteatomatous material. Or its edges may be denoted by cherry-red granulation tissue, which may attain to a considerable size. (See below.)

The perforation-sound on inflating the ears by Politzer's method, or on catheterizing, is in these cases frequently absent.

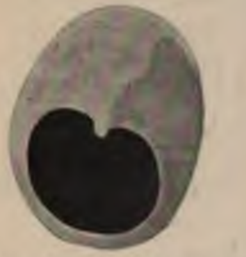

FIG. 167.—Reniform perforation of tympanic membrane.

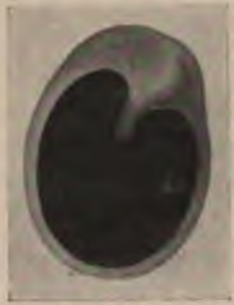

FIG. 168.—Total destruction of the vibrating membrane.

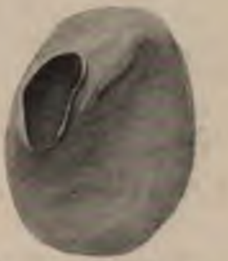

FIG. 169.—Perforation of the tympanic membrane in cholesteatoma. (See also Fig. 165.)

In any of the above varieties, **granulations** may be seen either inside the middle ear or protruding in clusters through the perforation. They may be so plentiful or so large as wholly to conceal the membrane, altogether filling up the lumen of the meatus. As we have seen, localized bulgings of Shrapnell's membrane may be mistaken for granulations, although the latter pit on pressure, and are more or less movable and very vascular. A granulation may increase so enormously in size as to fill the meatus and even protrude from its external orifice. When hypertrophied in this way, it obtains a covering of epithelium, and is generally spoken of as a *polypus*.

Bone-disease.—In any of the above varieties, also, the ossicles may be carious or necrosed. The incus, as we saw, is the most liable to necrosis; the malleus is occasionally, but the stapes is only rarely, diseased. In such cases careful probing through the perforation may result in the discovery of dead bone in the attic. Although, as we have seen, the chief focus of suppuration of the middle ear is in the mucous folds of the attic region, yet the term "**attic suppuration**" is reserved for a class of case, clinically well-defined, where caries or necrosis of the ossicles or walls of the attic, or indeed of the antrum and aditus, is discoverable, the typical site of the perforation in these cases being, as we have just seen, in Shrapnell's membrane or in the postero-superior meatal wall.

The presence of granulations and polypi always suggests diseased bone, and if, in spite of methodical treatment, with, perhaps, removal of the granulations and polypi, they keep on recurring, the suspicion of diseased bone becomes a certainty, and a mastoid operation is called for.

As a consequence of bone erosion, the chain of ossicles is apt to undergo destruction until, it may be, one or more of the individual bones is lost.

Inflation of the middle ear, save in certain cases of attic suppuration alluded to above, where the perforation is shut off from the general cavity of the tympanum, produces the usual whistling or blowing perforation-sound, and fluid may be heard bubbling as the stream of air passes through the tympanum.

Table showing the Complications and Sequelæ of Suppuration in the Middle Ear

From the *middle ear*, infection may spread :—

(*a*) By the veins of the tympanum direct to the jugular bulb, setting up septicæmia ; or septic thrombophlebitis of the bulb with symptoms like those of septic thrombosis of the lateral sinus (rare).

I. To the mastoid antrum and cells ; and thence—

(*a*) To the osseous tissue of the mastoid, causing necrosis, caries, or osteomyelitis ;

(*b*) To the outer cortex of the mastoid, causing mastoid abscess ;

(*c*) To the anterior cortex of the mastoid, causing abscess and bulging of the external auditory meatus, and at times abscess under the temporal muscle and sheath—temporal abscess—or over the external and upper aspect of the mastoid process.

(*d*) To the inner mastoid cortex, producing abscess within the mastoid sheath—Bezold's abscess.

(*e*) To the deep cervical region *viâ* the petrous process of the temporal bone, and thence to the pterygo-maxillary fossa and the pharynx (rare).

(*f*) *Viâ* the mastoid veins to the lateral sinus, causing sinus thrombosis, pneumonia, and pyæmia ;

(*g*) Directly to the groove of the lateral sinus, causing extradural abscess, and later, perhaps, sinus thrombosis, etc. ; or

(*h*) To the meninges or cerebellum, causing septic meningitis or cerebellar abscess.

II. To the cranial cavity, *viâ* the tegmen tympani and antri—the mastoid cells not being affected—and resulting in :

(*a*) Septic meningitis ;

(*b*) Extradural abscess ;

(*c*) Temporo-sphenoidal abscess.

III. To the labyrinth, setting up labyrinthitis, circumscribed or general, serous or purulent, and thence *viâ* the internal auditory meatus or aqueductus vestibuli to the meninges (abscess of the saccus ; meningitis) or cerebellum, or both.

These complications, it should be noted, may be consequent upon any of the varieties of suppuration of the middle ear, whether acute or chronic ; septic, cholesteatomatous, or tuberculous.

Each of these complications is dealt with in its place, and a consideration of the subject of suppuration of the middle ear involves the description of all of these, but at present we shall merely allude to what are generally agreed to be

Danger-Signals of the disease, which may appear at any moment, and which necessitate immediate operative treatment.

Pain.—The occurrence of pain is generally due to pus-retention. It is referred to the middle ear ; to the mastoid region ; to the occiput ; to the back of the neck ; or to the inferior maxilla, according to the site of the retention. The pain is often blended with

Headache, in the occipital or temporal regions ; or what is in reality much more frequent, in the frontal region. Quite commonly frontal headache of a low, unobtrusive character attends chronic middle ear suppuration, and as it is due to the general effects of simple septic absorption, it need not occasion alarm.

Pain or headache which is aggravated by lowering the head, or by pressing on the painful part, or by tapping the skull with the finger, should, however, always lead to operative exploration.

Vertigo occupies a similar position in the list of symptoms.

Aural vertigo is clear and definite in its character, and sometimes it is extremely disturbing. But there is no symptom to which patients pay less attention and forget so soon. And leading questions must be employed to elicit the fact of its occurrence in nearly all cases.

Definite vertigo in middle ear suppuration, often made worse by lowering the head, stooping, getting up in the morning, should also lead to an examination for the signs of labyrinthitis (q.v.), and in any case to exploration.

Bleeding from the ear; increase in discharge; sudden stoppage of discharge with the onset of pain; the presence of *granulations and polypi,* all indicate the need for prompt interference. And the same is true of *facial paralysis,* and of a *temperature of over 99° F.*

Progress.—Most cases of chronic suppuration of the middle ear tend to get well, especially when carefully attended to. Thus, one finds that when the disease begins in early life, it seldom continues until the patient is over middle age. But while this is true, there is, on the other hand, a considerable proportion of cases where suppuration continues with varying severity for many years; a smaller proportion which manifest a tendency to develop grave complications and come to operation; and finally a still smaller group that do develop grave complications, many of them dying in consequence.

It is the possibility of the fatal event that renders the disease even in apparently mild forms a source of anxiety.

Prognosis.—Favourable cases are those without any of the signs of severe local disease, such as granulations, polypi, or an inflamed membrane, in whom there is also no indication in pain, headache, or other threatening symptom of a tendency to complication. Favourable, also, is a readiness to react rapidly and considerably to local antiseptic treatment.

Treatment—Non-Operative.—The non-operative local treatment of chronic suppuration of the middle ear may be summed up in (*a*) drainage and (*b*) cleanliness.

All cases, at all ages should be examined for adenoids, and these, if present, even in small quantity, should be removed. The same caution applies to such nasal diseases as ozænatous rhinitis, or nasal sinus suppuration. Pyorrhœa, and other dental diseases should be removed or treated, and tonsils, if enlarged or septic, should be eradicated.

Locally, the meatus should be kept free of cotton-wool plugs, but it is difficult when the discharge is copious to induce patients to comply with this prohibition. All the same, these are the very cases where free exit for the discharge is important, and if the parts must be covered up for the sake of cleanliness, then a large dressing of gauze and wool, to include the whole of the auricle, should be applied and changed as often as necessary. Such cases are usually children, and owe the free outflow to adenoids.

When expert care can be regularly obtained, the chances of local treatment are much better than when the patient is left to treat the disease himself.

Syringing should not be practised. It is unnecessary, and not free from the risk of driving septic discharges into parts hitherto exempt from the disease.

The blowing in of dry boric or other powders is also obsolete.

To **Cleanse the Meatus and Middle Ear** the easiest plan is to mop the cavities out, under inspection if possible, with dry swabs of sterilized wool mounted on a probe. After drying out secretions, a few drops of (fresh) solution of hydrogen peroxide solution is dropped into the ear, and left there a few minutes, the patient meantime inclining the head so that the diseased ear is uppermost. When the foaming ceases, the ear is again mopped out with dry wool. Finally, antiseptic drops are inserted and allowed to remain in.

This toilette of the ear is performed twice or three times a day, according to the quantity of the discharge.

The antiseptic drops employed vary. The following are those most frequently used together with the indications for their respective use.

1. *For acute conditions;* when there is œdema and swelling of the membrane, or any tendency to pus retention or to irritation:

℞.	Glycerin. Ac. Carbol.,	ʒ ii. ss.
	Glycerin. ad	℥ i.—M.

2. *For ordinary antiseptic purposes:*

℞.	P. Ac. Boric.,	grs. viii.
	Spt. Vin. Rect.,	ʒ iv.
	Aq. ad	℥ i.—M.

or

℞. "Bacterol" in Aq. (1 to 250 or 1 to 500)
with or without alcohol.

3. *For œdematous granulations and polypi:*

℞.	Zinc. Chlorid.,	grs. iv.
	Glycerin.,	ʒ ii.
	Aq. ad	℥ i.—M.

N.B.—The zinc chloride precipitates albumens, and the canal must be carefully cleaned out when these drops are being used.

4. *For simple granulations*, for granulating surfaces, and also as the best drying and antiseptic agent,

Alcohol in strengths varying from 50 per cent. to 100 per cent. with water—the strength selected being the greatest the patient can tolerate. Alcohol causes burning and smarting, but to a variable extent.

5. *For cholesteatoma:*

℞.	P. Ac. Salicyl.,	grs. v.
	Spt. Vin. Rect.,	ʒ iv.
	Aq. ad	℥ i.—M.

N.B.—The surgeon must clean out the canal periodically when these drops are being used.

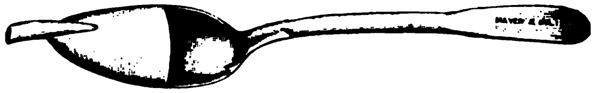

FIG. 170.—The author's Spoon-dropper for ear-drops.

INSTRUCTIONS TO PATIENTS FOR USE OF EAR-DROPS

1. Twist some absorbent cotton-wool round a bodkin, and dry out the ear with it.
2. Incline the head, or lie down, with the affected ear uppermost.
3. Pour in a few drops of "No. 1 Drops" (sol. hydrogen peroxid.) into the affected ear.
4. Leave the drops in the ear for five minutes, with the head still inclined.
5. Twist some absorbent cotton-wool round a bodkin, and

dry out the ear again with it. Do this thoroughly, renewing the wool until the ear is quite dry.

6. Pour in a few drops of " No. 2 Drops " into the affected ear.
7. Lie down for a few minutes with the affected ear uppermost.
8. Put a dry cotton-wool plug into the ear.
9. Remove this plug in ten minutes.

This should be repeated twice a day—night and morning.

In bed, lie with the affected ear DOWNWARDS.

Pain, or headache, or giddiness, or bleeding, or shivering, or feverishness, or increase in discharge, are all danger-signals, and should be at once reported.

Speaking generally, the greatest success follows the most assiduous cleanliness. Changes of drops from time to time are advisable.

A usual experience is to find the discharge becoming much less in first weeks of treatment, and then persisting unaltered for months. The remedy is perseverance with occasional changes of drops, so long as dangerous symptoms are not experienced.

If those danger-signs at any time appear, operation should, of course, at once be undertaken. (See p. 498.)

MASTOIDITIS

Some clinicians hold that suppuration in the tympanic cavity is always associated with suppuration in the mastoid cells, particularly in the *antrum*, the largest air-space in the process, and the one nearest the tympanum. The term " mastoiditis," however, is here applied only to those cases in which some overt and definite indications of mastoid osteitis exist. Mastoiditis may also occur independently of middle ear disease in tuberculous or syphilitic subjects, and in infants a primary osteomyelitis of the mastoid process has been described, but these varieties are very rare.

Otitic mastoiditis is due to a septic invasion of the mastoid diploë or pneumatic cells, producing osteitis with or without necrosis and the formation of a sequestrum or the production of caries.

In either case, if the escape of pus and other products of inflammation is not sufficiently free by the antrum-aditus-tympanum route, then the pus will burrow through the bone until it finds vent—

1. Through the external cortex of the mastoid process into the subperiosteal tissues above and behind the ear.
2. Through the internal cortex to form an abscess within the sheath of the sterno-mastoid muscle—*Bezold's abscess ;*
3. Through the roof of the antrum into the middle cranial fossa ; or,
4. Into the posterior cranial fossa, by rupture into the sulcus of the lateral sinus.

From the mastoid, infection may also reach the interior of the lateral sinus or of the brain.

Extension to the mastoid process and cells is uncommon in chronic suppuration, as might be expected from Cheatle's investigations (p. 478), but it is not unknown when an acute infection supervenes in the chronic disease. On the other hand, it is a common sequel of acute middle ear suppuration, and should be anticipated and watched for as long as acute symptoms continue.

Pathologically, acute mastoiditis must always, or nearly always, follow infection of the middle ear, but clinically, cases are met with where the middle ear disease is so trifling that the hearing is normal, and the membrane is of a natural appearance, and yet there is present mastoiditis with abscess formation. Indeed, this escape of the middle ear has been reported even in cases which have developed lateral sinus thrombosis or brain abscess.

Symptoms.—The classical symptoms of mastoiditis are as follows : *Pain,* referred to the mastoid region, frequently radiating over the head, generally worse at night, and accompanied by tenderness on pressure. In children nocturnal pain is of great significance. Mothers often tell how the child goes to bed free from pain, but wakes up about midnight screaming and crying with earache. This story, in the absence of signs of pus-retention in the tympanum, should always arouse suspicion of mastoid involvement.

Tenderness on pressure over the mastoid region is also a sign of much importance, and must be sought for with great care. The typical sites where digital pressure should be made are :

1. Over the antrum, pressing deeply backwards and inwards behind the auricle at a spot corresponding to the postero-superior quadrant of the meatal wall ;

2. About the centre of the process;
3. At the margin of the hairy scalp on a level with the external meatus, corresponding roughly with the exit of the mastoid emissary vein; and,
4. Over the tip of the process, which, it should be remembered, lies well forward under cover of the concha.

In eliciting this symptom, the finger should be applied in such a way as not to displace the auricle or the cartilaginous meatus, for, in furunculosis of the canal, the slightest movement of auricle or pinna is extremely painful, and may give rise to the erroneous impression that mastoid osteitis is also present.

After palpating the suspected mastoid, the other side should also be examined in the same way, as some people are very sensitive to pressure in this region, and the tenderness they feel when the part is palpated may be no more than the normal sensation.

In addition to tenderness, in most cases of purulent mastoiditis, even at a very early stage, *œdema of the soft tissues over the mastoid and in the scalp* behind the mastoid will be found on examination. This symptom may be regarded as a very reliable indication of deep pus in the bone either of the mastoid or of the occiput. It is found readily, and is very obvious when the lateral sinus is the seat of thrombosis, in which case the bogginess will be found to extend posteriorly towards the occiput. But even in early and simple mastoid suppuration, and long before the soft tissues are actually invaded, pitting on pressure can be produced over the mastoid process or along the edge of the hairy scalp, and when found, it may, we believe, be always regarded as a reliable indication of pus in the mastoid process, or of lateral sinus thrombosis. Provided, of course, that the case is not one of furunculosis or of œdema from Bright's Disease, or some other local or general condition, such as adenitis, etc.

In order to produce the pitting, the finger must be applied with considerable force, and the pressure of the soft parts against the bone is to be kept up for from 15 to 30 seconds. Pressure of this kind does not induce pitting in health.

The *temperature* in uncomplicated mastoiditis seldom or never rises above 100° F. In children, exceptions to this rule are often found, but in adults it is rare to find any exception. So that if in an adult the temperature exceeds this height, some other complication, the most usual being lateral sinus thrombosis, should be looked for.

The varieties of **Abscess of the Soft Tissues** due to an extension from the middle ear and mastoid cavities are (1) the temporal abscess; (2) the post-auricular, or mastoid abscess; (3) Bezold's abscess, in the sheath of the sterno-mastoid; and (4) one of great rarity, the sub-petrous or pharyngeal abscess. Of the four, the first is the commonest.

Temporal Abscess.—In this variety the abscess is situated within the sheath of the temporal muscle; that is to say, it is confined behind and below by the attachment of the temporal fascia to the linea temporalis. There are two routes by which the pus reaches this situation. The first is from the middle ear along the roof of the meatus burrowing between the bone and the soft tissues, until it reaches the orifice of the bony meatus, when it makes its way upwards and backwards in the temporal fossa above and behind the auricle—in this case the mastoid process may not be involved in the disease at all. Secondly, a temporal abscess results when the pus from the mastoid cells breaks through the outer cortex of the process *above* the linea temporalis, above and behind the auricle—a by no means rare development when air-cells are plentiful and approach the outer cortex closely in the neighbourhood of the root of the zygoma.

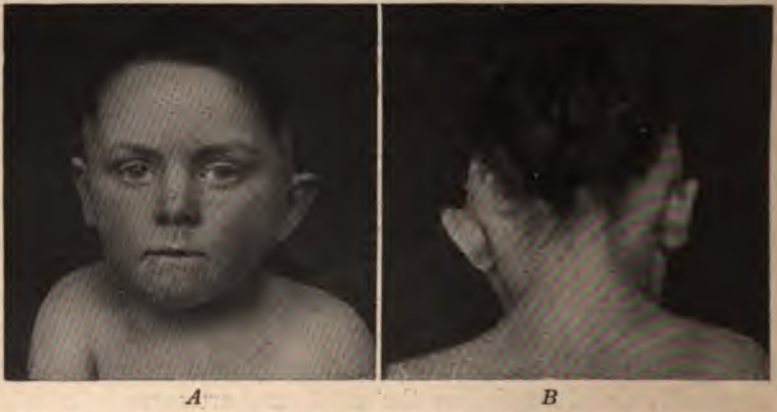

FIG. 171.—*A*—Anterior, and *B*—Posterior view of a child with abscess of the temporal fossa due to mastoid suppuration, showing the typical protrusion and depression of the auricle. Hair partly shaved.

The *symptoms* are those of abscess of the soft parts. The auricle is generally very prominent compared with the healthy side, and appears to be driven in a direction downward and outward. (Fig. 171.) The whole of the temporal and, to some extent, the mastoid regions are tense, swollen, tender, and œdematous, but the tip of the mastoid process is often quite palpable, and free from any abnormal sensitiveness.

When the pus has tracked along the meatal roof, examination with the speculum will show the meatal lumen encroached upon by its swollen and sagging upper wall.

Post-Auricular or Mastoid Abscess arises when pus finds a way out on to the subcutaneous surface of the process. The usual route is by direct breach of the bone of the outer cortex, by which a fistula is formed between the cells filled with pus and granulations, and the periosteal and other tissues over the mastoid process. The fistula may appear almost anywhere, but it is most commonly found in the anterior and upper quadrant.

The *symptoms* are those of abscess of the soft parts, and call for no particular description.

Diagnosis.—The disease with which mastoid abscess is most liable to be confused is furunculosis of the meatus. (See p. 446.)

Rarely, the post-auricular lymphatic gland is the seat of adenitis and abscess, from scalp infection. In this case, the original site of the infection is usually obvious, while the middle ear and membrane are normal.

Cysts, lipomata, tuberculous abscesses in this region are easily differentiated from the acute abscess.

Bezold's Abscess.—In this variety the pus emerges from the mastoid in the neighbourhood of the tip—usually at its inner aspect—and within the sheath of the sterno-mastoid muscle.

Symptoms.—The pain from the inflammatory reaction in the upper section of the sterno-mastoid muscle interferes with the action of the muscle, and the patient is compelled to hold the head in a characteristically stiff-necked attitude, the head being inclined towards the diseased side. On examination, the neck below and behind the mastoid process and auricle is seen to be the seat of a phlegmonous swelling and infiltration, pitting on pressure and very tender.

The **pharyngeal abscess** is a rarity. It is due to the extension towards the pharynx of a deep cervical cellulitis close under the base of the skull, the pus usually appearing in the peritonsillar region.

Symptoms.—In addition to the usual constitutional signs of deep pus, the movements of the jaw are incommoded, the mouth being fixed in a half-open position. There is pain referred to the lower jaw or temple, with œdema of these regions, while examination of the pharynx reveals swelling in the soft palate above and external to the tonsil.

(The intracranial extensions and complications of middle ear suppuration are described below. In the meantime, it is convenient to pass here to the operative treatment of the disease as it affects the ear and its external neighbourhood.)

THE MASTOID OPERATIONS

(Before proceeding to describe these operations, it is necessary to state that while most operating otologists agree as to most of the general principles in the operations, great individual variety is noticeable in detail. Consequently, the writer is perforce compelled to limit himself generally to his own methods and practice.)

THE CORTICAL MASTOID OPERATION (SCHWARTZE'S OPERATION)

Description.—This consists in opening and draining the mastoid process, antrum, and cells, the middle ear and its contents being left alone.

Indications.

1. Purulent mastoiditis with or without extension of the disease of the soft parts.
2. Persistence of discharge after acute suppuration of the middle ear for six weeks or longer, without mastoiditis, provided that simple treatment (see p. 490) has been carefully carried out.

Operation.—A post-aural incision is made, curving from the junction of the upper surface of the pinna with the skin of the cranium downwards in a crescentic curve along the middle of the mastoid region to the tip of the process. (Fig. 173.) The knife is carried down to, but not through the temporal fascia above, and to and through the soft tissues and periosteum over the mastoid below, the incision being deepened here until the surface of the bone is reached.

An elevator is now introduced, and the mastoid process is exposed by elevation of periosteum and soft tissues, from the external bony meatus in front, to the tip below, and the posterior limits of the mastoid behind. Next, the mastoid retractor is fixed in position. (See Fig. 173.)

Then, having formally recognized the landmarks, of which the external bony meatus is the most important, the mastoid cells are exposed by elevating the outer cortex of the bone with gouge and mallet, with cutting bone forceps, or with osteotribe (burr), according to the nature of the mastoid and the type of the disease present.

The cells thus opened, if filled with granulations, are entirely cleared by means of a sharp curette under the guidance of Grant's probe; and this opening up of cells and their clearance is continued until the whole of the obviously diseased areas and extensions have been reached and thoroughly opened up.

The rule is to follow the disease wherever it leads us. This may lead to an exposed lateral sinus, or dura mater, often covered with granulations, but these granulations it is wiser not to disturb, unless symptoms indicating the involvement of these structures have been present.

Finally, all the extensions, major and minor, having been tracked to their ultimate recesses, the mastoid antrum is found and freely laid open from the posterior or mastoid side, granulations here being also scooped out. (Fig. 174.)

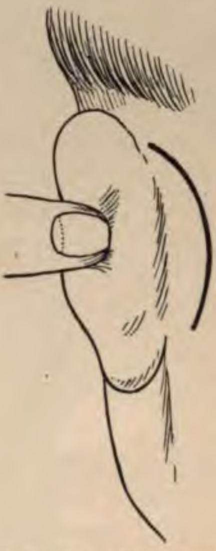

FIG. 172.—Post-aural incision for the Cortical Mastoid (Schwartze's) Operation.

The bony posterior meatal wall is now nibbled partly away; the cavities are freely treated with bismuth-iodoform-paraffin-paste (" B.I.P.P.") (p. 356) and lightly packed with gauze, and the upper section of the skin-wound is closed by sutures, the lower end being left open for drainage. A dry dressing is applied.

Before dressings are applied, it is advisable to make an incision through the membrana tympani as in paracentesis, if this has not already been done, in order to obtain free drainage of the tympanic cavity.

Difficulties and Dangers.—The most usual difficulty is due to swelling of the soft parts whereby one is apt to lose the sense of direction. The chief landmark is the external bony meatus.

The disease may have led to a denudation of the lateral sinus, and as it is then often covered with granulations, its presence is not revealed to the eye, but only to the probe. If the sinus is inadvertently opened, free bleeding at once takes place. (See p. 538.)

After-treatment.—After all operations on the ear, the patient should, if possible, lie upon the affected side. (Fig. 176.) The wound is dressed daily, boric acid fomentations being substituted for the dry dressing of the first twenty-four hours.

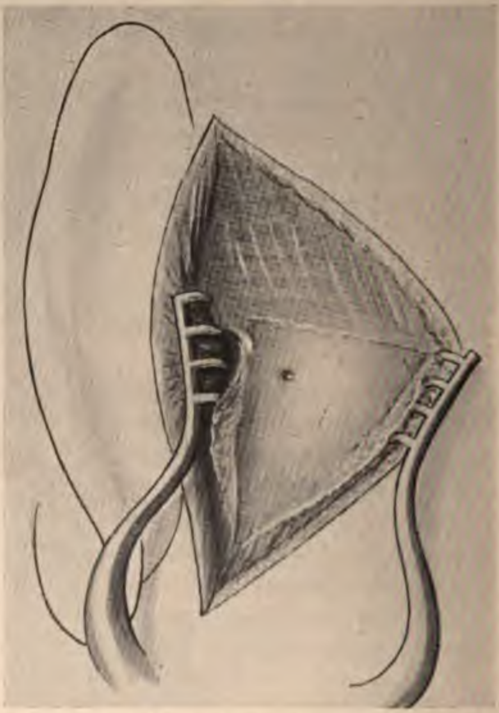

FIG. 173.—The Cortical Mastoid Operation (Schwartze's Operation).
(1) Exposure of the surface of the mastoid process; showing a fistulous opening The usual œdema of the soft tissues is not indicated.

The case is closely watched for the development of symptoms indicating danger, and the temperature is taken four-hourly. Persistent pain or headache, spontaneous nystagmus, and cervical rigidity should be looked for daily, while the condition of the wound is also being watched for the occurrence of local complications of a septic character, as these are not unusual in the presence of the virulent infection characteristic of many of these cases.

Results.—Complications apart, the results of the cortical mastoid operation in suitable cases are excellent. In from a fortnight to six weeks the post-aural wound closes; the meatal discharge dries up; and the middle ear not having been interfered with by operation, and but little by disease, the hearing is, to a great extent, or entirely, restored. In a word, the operation saves the hearing.

After the wound is healed, and all aural discharges stopped, the nose and naso-pharynx should receive attention—adenoids, if present, being removed to prevent a recurrence of the ear suppuration.

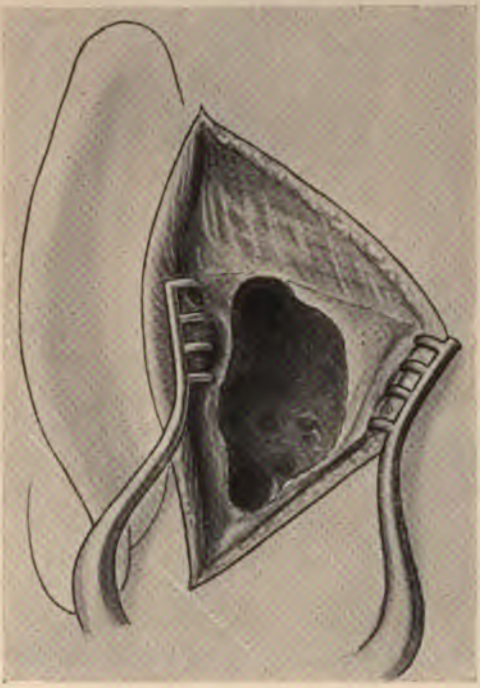

FIG. 174.—The Cortical Mastoid Operation.
(2) A cellular mastoid laid open; granulations and pus removed, and antrum opened for drainage.

The radical mastoid operation will have to be performed after the cortical operation if complications, apart from local infections, arise during convalescence; or if the meatal discharge continues unabated after the healing of the post-aural wound.

The *Abscesses in the Soft Parts* may be opened in the course of the cortical mastoid operation, if there is, e.g., *retro-auricular mastoid abscess;* while the *Bezold Abscess* may be opened from the lower end of the mastoid incision after the cells at the tip have been freely opened up, and perhaps the fistula leading to the abscess discovered. A separate incision in the neck may be, but seldom is, necessary as a counter-opening.

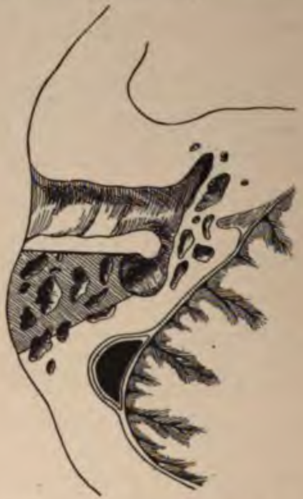

FIG. 175.—Cortical Mastoid Operation. Coronal section (diagrammatic) showing external auditory meatus; a cellular mastoid process; the lateral sinus (black) and the cerebellum. The shaded area represents the bone removed in the cortical operation. (From Laurens, *Chirurgie Oto-rhino-laryngologique.*)

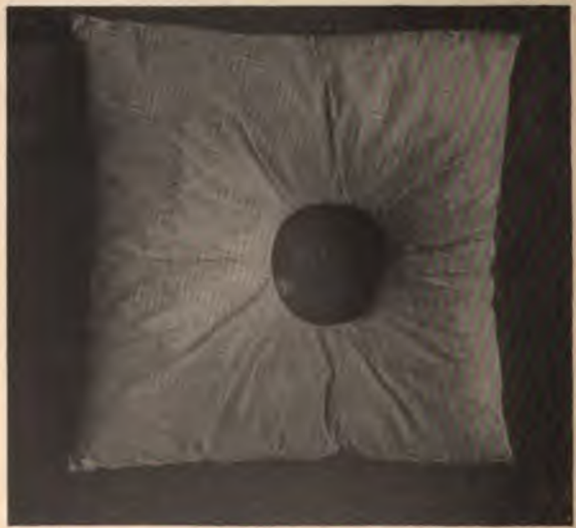

FIG. 176.—The Author's Mastoid Pillow; to enable the patient to lie with the affected ear downwards, in order to favour drainage.

Temporal Abscess is opened by a vertical incision through the hairy scalp above the auricle, and if the pus has tracked out along the roof of the meatus, a counter-opening made here will obviate any necessity of operation on the mastoid cells—unless, of course, there is any reason to believe that they are involved.

Naturally, a temporal abscess which is due to fistula opening from the mastoid cells above the linea temporalis calls for operation on the bone.

Otogenic Pharyngeal Abscess may be opened through the mouth, the incision being made over the most prominent part of the swelling in the throat.

THE MODIFIED MASTOID OPERATION

(The Conservative Mastoid Operation)

Description.—The opening of the mastoid antrum for drainage, the tympanum not being opened up.

Indication.—Persistence of suppuration of the middle ear in spite of free drainage through the membrane, without signs of mastoiditis.*

Contra-Indication.—The usefulness of this operation, like that of the cortical operation, of which it is a modification, is in the author's belief confined to cases where the discharge is of brief duration—from six weeks to three months.

It is inapplicable to cases with caries of the ossicles, or with cholesteatoma, granulation tissue or polypus formation in the tympanic cavity, and *a fortiori* to cases in which serious complications threaten. For all of these, the radical mastoid is necessary.

Operation.—Incision as in the last, or close in the auriculo-mastoid angle.

After the bone is cleared, the mastoid process is opened with gouge and mallet, and the cavity thus formed is deepened in a slightly upward and forward direction towards the mastoid antrum, which lies about an inch, more or less, deep to the supra-meatal spine, or to Macewen's triangle. (See Fig. 178.)

The mastoid antrum having been reached, free drainage of that cavity is ensured by a sufficiently large opening in its posterior and external walls, and any cells or canals leading from it are followed up and laid open also.

The posterior wall of the bony meatus is partly removed, and it is then shaved down with the chisel until the floors of the meatus and the cavity in the bone are approximately on a level. (Fig. 182.) But "the bridge" is not broken down; the aditus is not exposed; the membrane, ossicles, and tympanic cavity are left untouched.

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* Many operators do not resort to this modification unless signs of mastoiditis are present.

The bone cavity may be grafted as in performing the radical mastoid operation (p. 513), but C. J. Heath cuts a meatal flap (p. 506), which is laid back into the bone cavity as in the radical operation.

The cavity is then treated with B.I.P.P., and the post-aural wound is completely closed as in the radical operation.

Difficulties and Dangers.—As in all mastoid operations, one avoids exposing the lateral sinus and the dura. It is, indeed, often impossible to avoid exposing either or both of these structures. (See later, p. 509.)

The facial nerve is not endangered in either of the two operations we have just described.

After-Treatment.—As in the last.

Results.—Favourable cases heal up in from two to three weeks, and the meatal discharge also comes to a standstill.

Many of the details of this operation, combining as it does some of the features of the cortical and some of the radical operation, have been worked out by C. J. Heath.

The writer's experience, which he believes to be general, shows that, apart from early acute cases for which simple myringotomy, or it may be the cortical mastoid operation described above (p. 498), is suitable, the modified or conservative mastoid operation as detailed by Heath is disappointing. Its utility, in point of fact, is confined to the small class of case, usually cholesteatomatus, in which tympanic disease is slight or absent. As it does not open up the tympanum, it does not reveal what is the seat of the disease in the great majority of chronic cases. Unless most cautiously adopted, therefore, it will not only expose the patient to the needless risk of two major operations, but it may also lead to a serious and even to a fatal waste of time.

If used for early acute cases, it will be largely successful. But then, as we have seen, equally brilliant results admittedly follow simple incision of the membrane on the one hand, or the simple cortical mastoid operation on the other.

Finally, when the radical mastoid operation, about to be described, is necessary, to stop short in the course of that operation and to content oneself with a conservative procedure, is to expose the patient to a continuance of the risks for the removal of which the operation was undertaken.

THE RADICAL MASTOID OPERATION

Description.—The name given to this operation is unfortunate. It is, in fact, an antro-tympanic partial evisceration; there is nothing " radical " about it; and in most cases, the operation is only " mastoid " in the sense that it is performed through a small part of the mastoid process.

The operation consists in throwing the antrum and tympanum into one cavity; in clearing out all the tympanic contents except the stapes; and in removing as much of the diseased bone as possible.

Indications.—The presence of any one of such symptoms as pain; persistent headache; vertigo; recurrent polypi and granulations; pyrexia, i.e., evening temperatures of over 99° F.; facial paralysis; and of course, of the signs of the graver complications.

It may also be performed if the cortical or the modified operation fails to cure the discharge.

Acute phenomena in a chronic suppuration call for the radical operation. This is the reason why one must always, when confronted with mastoid abscess, make certain of the date when the discharge from the meatus first commenced. If it has been in existence more than three months; or if it has occurred frequently before with intervals of abeyance of discharge, then the radical operation is necessary.

Otherwise the cortical operation is sufficient.

Mastoid Operations in the early stages of Middle Ear Suppuration.—The treatment of suppuration of the ear should be devoted to cutting short the discharge at the earliest possible moment.

First of all, if the drainage through the membrane is not free, myringotomy should be performed (p. 476).

2. If pain and temperature or other grave signs (see p. 494) persist after this operation, the cortical mastoid operation is indicated (p. 498).

3. If such grave signs being absent, the discharge continues for over a week, the throat should be cleared of adenoids and tonsils if they are present.

4. Tonsils and adenoids not being present, or having been removed, a month or six weeks may be spent in meatal treatment, so long as urgent symptoms do not appear.

5. If, however, in spite of the meatal treatment, the discharge

continues for a month or longer, grave signs being absent, the mastoid antrum should be opened and drained by the cortical operation, as described above.

6. If, in spite of this drainage of the antrum, the discharge still continues, then the presence of tympanic bone-disease, or of cholesteatoma in the tympanum may be taken for granted, and only the radical operation can cope with those conditions.

But, after the cortical operation, *unless deafness is severe or urgent signs are present*, meatal treatment should be continued so long as any hope exists of cure, the reason being that the exenteration of the tympanum in the radical operation is often followed by considerable loss of hearing. If, however, grave symptoms exist, the radical operation must be at once performed, regardless of the hearing.

The above caution as to the radical operation is of less importance if the other ear is healthy and its hearing good.

These rules apply with particular force to children, in whom the great majority of acute cases, and 50 per cent. of chronic cases (Kerr Love) get well with simple meatal treatment.

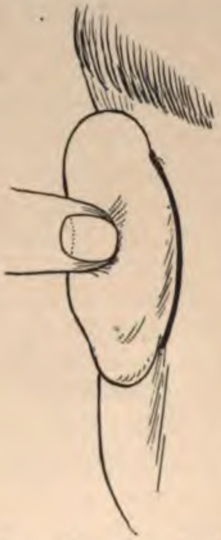

FIG. 177.—Post-aural incision for the Radical Mastoid Operation.

Contra-Indications.—The usual contra-indications of major surgical operations. If acute labyrinth symptoms are present, the operation should be postponed until they disappear. (See p. 522.)

Operation.—The skin incision runs close to the auricle in the auricle-mastoid angle, and is carried from the upper attachment of the auricle as far down as the tip of the mastoid process. (Fig 177.) The bone is cleared as in the preceding operations, but in addition, the skin lining of the bony meatus—the "membranous" meatus—is separated from the bony meatus along its upper, its posterior, and part of its inferior walls, and split with a knife in such a way as to form a "flap" for the partial lining of the cavity in the bone about to be formed.

Cutting the Meatal Flap.—There are many varieties of meatal flap; the following is as good as any. An incision is made in the roof of the meatus from within outwards as far as the concha, being carried down to the bone. From the outer end of this incision, and at right angles to it, a second incision is made through the posterior wall of the meatus, including, perhaps, part of the concha. These incisions are made with the meatus in *situ*, under inspection from in front and from behind (through the mastoid wound) alternately, the auricle being laid forward and turned back according to necessity. When the incisions are completed, the auricle is held strongly forward, and the flap defined by these incisions is cleared and separated from the bony meatus. The outer or conchal incision generally severs the posterior auricular artery, which has to be secured. The subcutaneous tissue of the flap, which is generally thick and abundant, is removed by dissection, in the course of which some of the conchal cartilage often needs removal. There is now left a tongue of skin which, after bone operation is completed, is laid back into the bone cavity, and secured to the mastoid periosteum with one or two catgut sutures. (When a graft is applied, the flap cut as above is entirely removed).

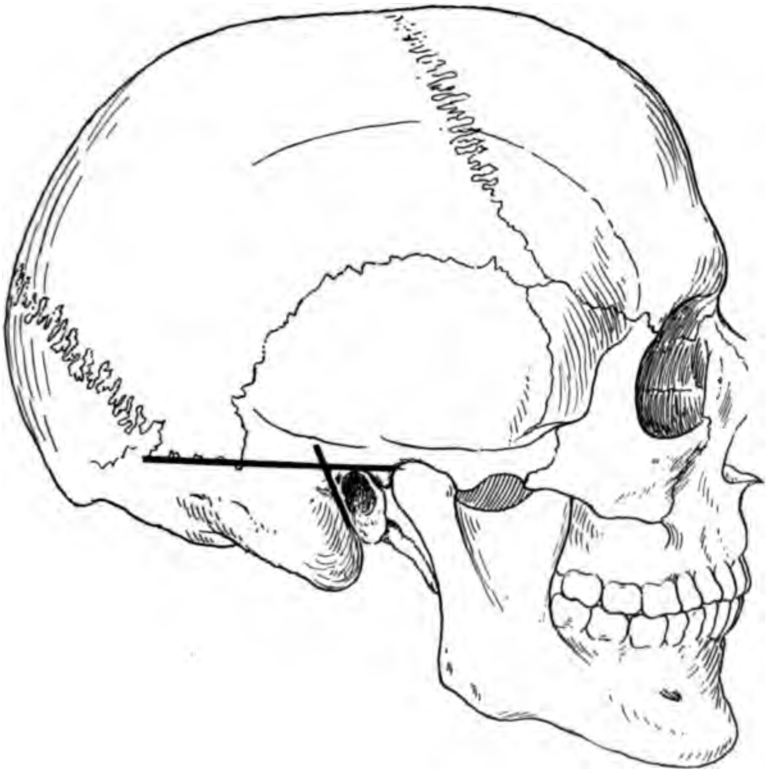

FIG. 178.—Skull showing Macewen's triangle, the landmark for the mastoid antrum.

Whichever plan is to be adopted, whether flap or graft, we are now left with the mastoid and the adjoining walls of the bony meatus entirely denuded of soft parts, while the auricle is displaced forward out of the way. A sterilized strip of gauze or bandage, inserted through the natural orifice of the meatus at the auricle, and through the artificial opening or "window" we have just made in the split meatal posterior wall, is given to an assistant, in order that he may hold the whole auricle forward out of the surgeon's way.

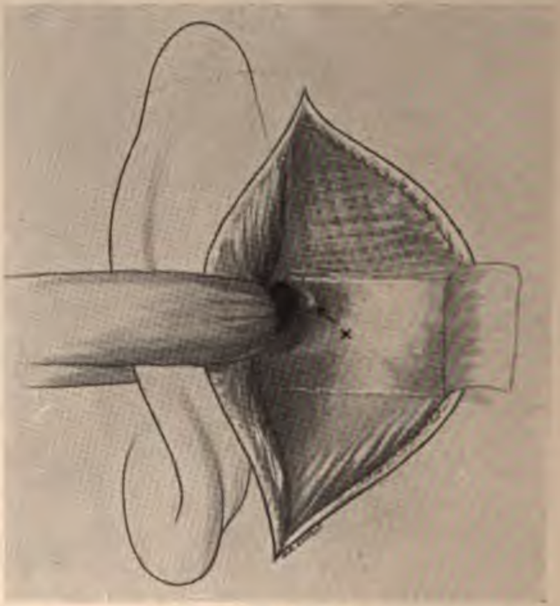

FIG. 179.—The Radical Mastoid Operation (1).

The exposure of the surface of the bone.

The arrow points to the supra-meatal spine; and the cross indicates where the bone is opened in beginning the tunnel-excavation.

All bleeding points having been caught up with forceps so as to secure a dry wound, the operation on the bone is now begun.

The first step consists in obtaining an entrance to the mastoid antrum through the upper and anterior region of the mastoid process. The surface landmark of the antrum is the "supra-meatal spine," a short process of bone projecting from the surface at the outer edge of the postero-superior quadrant of the bony meatus. (Fig. 179.) As this spine is frequently absent, perhaps what is known as "Macewen's triangle" is a more reliable guide. This is a triangular area formed by the intersection of lines drawn tangential to the posterior and superior meatal walls respectively, by the postero-superior segment of the bony meatus. g. 178.) The mastoid antrum lies about ¾″ deep to this a.

(The situation of the antrum relative to the external bony atus varies with the age; at birth and in infancy it lies erior to the meatus; in childhood it lies a little further hind; in adult life it lies still further behind, and at a lower el. See Fig. 165.)

With gouge and mallet, a tunnel is excavated by removing he bone of the upper anterior region of the mastoid process, nd by deepening this excavation inwards and slightly forwards in a direction roughly parallel with the bony external meatus. The excavation is continued until the mastoid antrum is opened into from behind. (Fig. 181.)

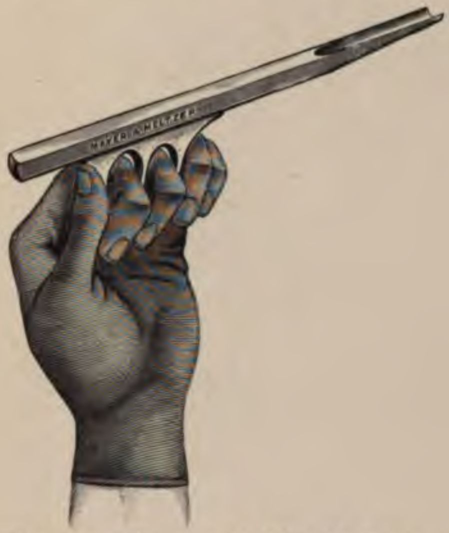

FIG. 180.—The author's Mastoid Gouge.

In carrying out this step, two dangers are to be guarded against, the exposure and wounding (1) of the lateral sinus; and (2) of the dura mater of the middle fossa.

The lateral sinus often approaches quite close to the bony meatus, and its exposure may be unavoidable. If exposed without being wounded, the operation is not inconvenienced. If it is, however, wounded, the sinus must be packed with gauze (see p. 538) to stop the very free hæmorrhage which follows, and this packing may hinder the completion of the operation.

Exposure of the dura mater is of no consequence unless it is wounded, but in any case, the accident need not interfere with the mastoid operation. The landmark of the dura of the middle fossa is the linea temporalis and the attachment of the temporal fascia, but if, as is commonly the case, the antrum lies high, this level must be transgressed by the operator in the deeper parts of the bone cavity.

If the dura is wounded, at the completion of the mastoid operation, the dural wound, if small, should be freely enlarged, and drainage provided for cerebro-spinal fluid by inserting the tail end of a fine strip of iodoform gauze between the lips of the dural wound and into the cerebral substance if necessary. If this is done, the accident will entail no serious consequences.

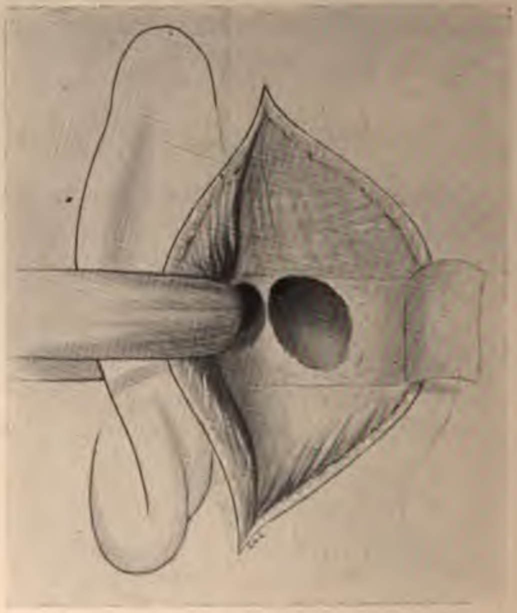

FIG. 181.—The Radical Mastoid Operation (2).
The tunnel excavation leading to the mastoid antrum.

We resume now the description of the radical mastoid operation.

The antrum having been opened, the next step is the removal of the "bridge" of bone overlying the aditus between the tympanum and the opened antrum. (Fig. 182.) This must be done with a due regard to the position of the facial nerve, which runs vertically downward from a point 2 mm. inferior to the floor of the aditus in the plane corresponding to the outer lip of the posterior wall of the bony meatus. (Fig. 195.)

The removal of the "bridge" is effected by enlarging the antrum opening with a fine gouge in a forward direction, and then by direct attack upon what remains of the " bridge " with a sharp chisel and mallet from without. If the operator is careful, he will at this stage insert from behind or from in front, Grant's mastoid probe, as this instrument protects the facial nerve. The breaking of the last piece of the " bridge " is effected by light tapping on the chisel, and when it takes place, the sound and feel of the fracture is unmistakable. (Fig. 183.)

The antrum and aditus are now exposed, and the next stage consists in clearing the tympanic cavity of what remains of membrane and malleus and incus together with granulations and polypi, if there are any present. At the same time, the roof of the antro-tympanic cavities is fully exposed by gouging away its overhanging outer wall, while " the facial ridge," that is to say what remains of the posterior bony wall of the meatus, is planed down with a sharp chisel. Finally, any deep mastoid cells which may be found are followed up and thoroughly laid open, all granulations being curetted away.

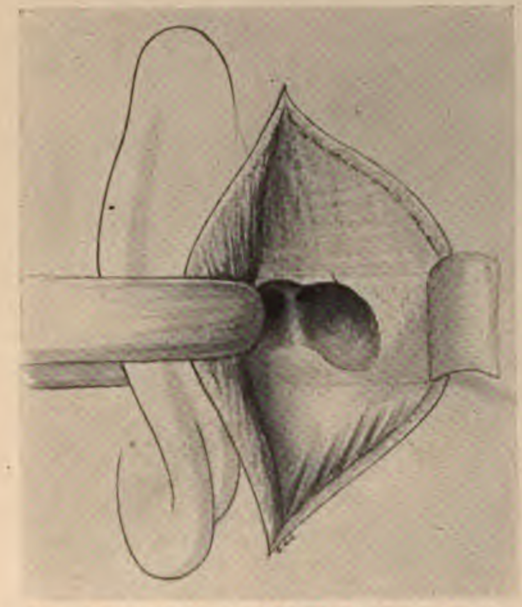

FIG. 182.—The Radical Mastoid Operation (3).
The tunnel excavation has reached the antrum, while the " bridge " is partly demolished.

The inner antro-tympanic wall is next cleared of granulations and swollen mucous lining by means of fine curettes, and a careful search is made for indications of extension of the disease-process in this inner wall towards the labyrinth by way of (1) the external semicircular canal which is easily visible as a smooth white bulging and pillow-like prominence on the floor or the inner wall of the aditus; or (2) by way of the vestibule in the neighbourhood of the oval window. (Fig. 184.) A fistula in the external semicircular canal leading to the interior of the labyrinth is denoted by the presence of a small oval opening in the bone occupied by a granulation, which the operator will do well to let alone. Fistulous openings about the oval window are less easily discovered, as it is a difficult region to inspect. To do so, the patient's face should be turned round so as to bring the occiput more on to the table.

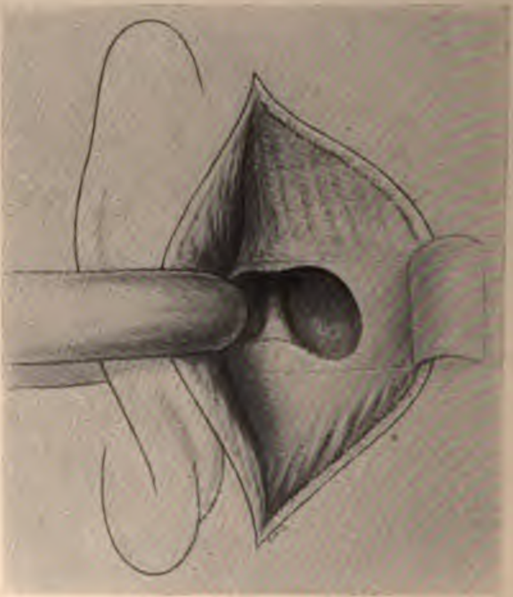

FIG. 183.—The Radical Mastoid Operation (4).

The "bridge" has been broken down, revealing the aditus ad antrum with the prominence of the external semicircular canal.

Another favourite site for caries and granulations is the sinus tympanicus, a *cul-de-sac* in the postero-external wall of the tympanic cavity, behind the membrane, and here the facial nerve may be exposed by the disease. Curetting this region must therefore be cautiously carried out.

After the roof and all the walls of the bony cavities have been carefully inspected, and have been interrogated with the probe in order to discover all extensions of disease towards cerebrum, cerebellum, or lateral sinus, the tympanic orifice of the Eustachian tube is curetted free of granulations.

Finally, a large square skin-graft having been cut from the thigh, it is floated in on normal saline into the bone cavities, being laid against all the bony surfaces that have been formed or exposed in the course of our manipulations. (Fig. 185.)

Then the cavities are treated with B.I.P.P. (p. 356), and filled with fine iodoform gauze, packed gently in so as to retain the graft in position, its end being brought out at the orifice of the meatus in the auricle, while the graft is doubled over the packing which it encloses like a pudding-bag. (Fig. 186.) The auricle is then replaced, and the outer tail of the graft, or if necessary, a second graft may be inserted in such a position as to cover the raw edge of the cut membranous meatus at its passage into the concha. (Fig. 187.) This is useful in preventing the formation of exuberant granulations at this place. The skin wound is united with six or seven interrupted sutures, and a dry dressing applied.

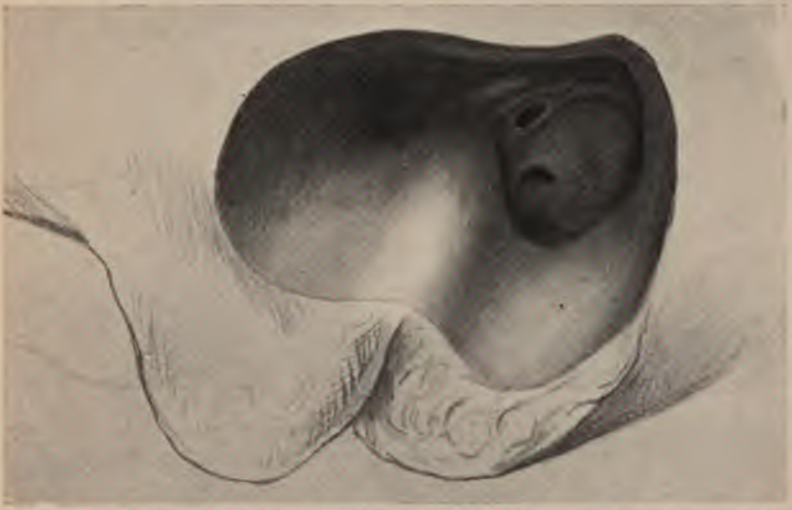

FIG. 184.—The radical mastoid bone cavity (diagrammatic) showing the antro-tympanic cavities with the prominence of the external semicircular canal; the facial canal; the oval window; and the round window. (In order to display these features the medial tympanic wall has been brought into the sagittal plane and enlarged.)

After-Treatment.—The outer dressing is changed daily. But the packing is left for four or five days. It can then be removed without disturbing the graft, and should not be re-inserted.

During convalescence, the patient should be watched carefully for complications,—pain, continual headache, vertigo, sickness, or pyrexia, being the indications of danger. Locally, the signs of septic infection of the wound are met with by suitable treatment, but such septic infection is less frequent since B.I.P.P. was introduced.

Results.—In so far as immediate danger to life is concerned, the radical mastoid operation in uncomplicated cases is practically free from risk, the mortality being only about 0.5 per cent. (J. S. Fraser).

The outer skin wound heals in about a week. The bone cavity takes from three weeks to six months to become epithelialized and dry in favourable cases. In less favourable cases, about 25 per cent. of the whole, the discharge continues, or recurs from time to time for an indefinite period.

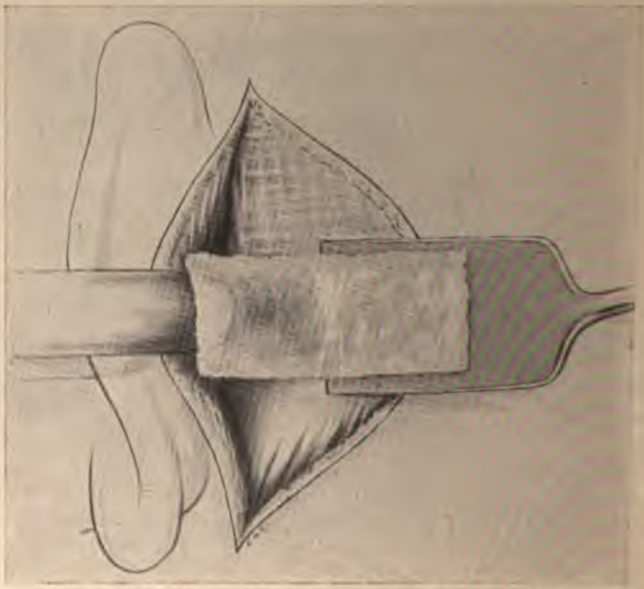

FIG. 185.—The Radical Mastoid Operation (5).
The application of the skin-graft.

But the object of the operation is in most cases obtained apart from cessation of discharge. For, it should be remembered that the "radical mastoid" (so-called) is an exploratory procedure, and that it tends to prevent the further extension of the bone disease.

The best results are obtained when the surgeon himself is able to carry out the after-treatment, and to maintain asepsis. This last may, with care, be managed for an indefinite period, unless in those cases where infection reaches the cavities from the naso-pharynx, as in people subject to "colds."

Daily packing of the bone-wound is useless and obsolete, and, indeed, the less the interior of the ear-wound is touched, even aseptically, the better.

Re-opening.—The occurrence of signs of complication during convalescence may necessitate re-opening, and the same may be required if the discharge is very free and continues without abatement, or if granulations and polypi re-form.

Frequently, a re-opening, curetting, and re-grafting will rapidly lead to complete cure.

The reason for the uncertainty attaching to the question of the cure of the discharge is that in many cases it is impossible to eradicate the disease without sacrificing either the labyrinth or the facial nerve, or both. And this is, naturally, out of the question.

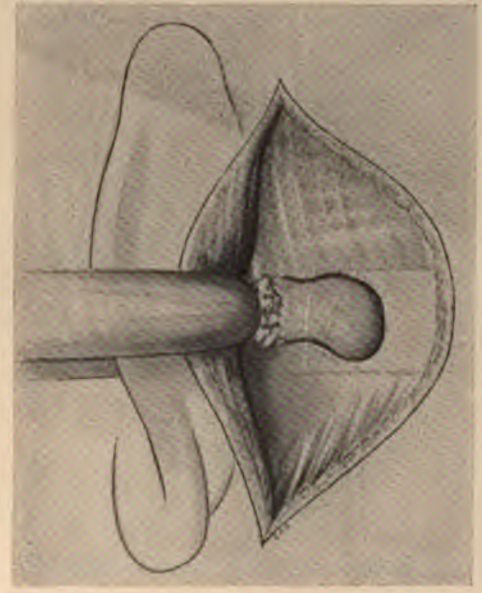

FIG. 186.—The Radical Mastoid Operation (6).

The skin-graft in the bone cavity is held in position by a gauze packing. Note how the graft is brought over the packing like a pudding-bag, so as to line the wound in the soft tissues when the auricle is replaced.

Far from being "radical," the operation is essentially a compromise.

The *Effect on the Hearing* of the radical mastoid operation varies. As a rough rule, it may be stated that if the hearing is seriously impaired before the operation, it will probably be improved after; while on the other hand, if the hearing before the operation is good, it will probably be not so good after the operation.

One of the advantages claimed for the skin-graft over the meatal flap as a method of lining the cavities is that the hearing is less interfered with by the fine thin scar of the graft..

If after the radical mastoid the whisper can be heard at from three to five feet, the functional result is satisfactory.

Mastoid Operations in Cholesteatomatous Disease follow the general principles we have just laid down; but, as may be gathered from what has been said on the pathology of cholesteatoma (p 480), we frequently find on opening the bone that the disease itself has already produced an excavation of the same kind as that which the operator seeks to effect. For that reason, a mastoid operation may be a comparatively trivial procedure in this disease.

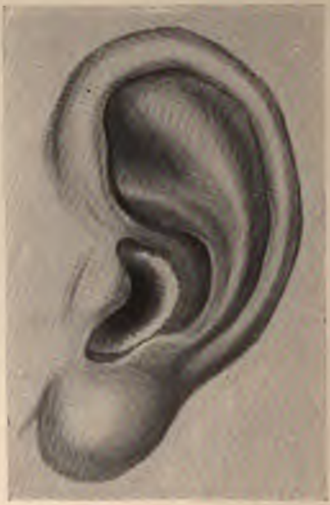

FIG. 187.—The Radical Mastoid Operation (7).

The auricle replaced and the edge of the graft laid round the cut margin of the meatus like a fringe in the concha.

Again, we not infrequently discover on reaching the antrum that the disease is confined to this region, or it may be to the adjoining aditus, the ossicles and middle ear being free of the disease (p. 481). In such cases, the tympanic membrane, the ossicles and the attic should not be interfered with, and although the " bridge " may be removed, care should be taken not to displace the short process of the incus from its seat on the floor of the aditus.

In contrast with such cases of partial and limited destruction, however, we find sometimes on the other hand that the cholesteatomatous growth has removed the protective bone-covering from important structures and organs; the labyrinth-wall may be eroded; the facial nerve extensively exposed; the lateral sinus and dura mater laid bare. And all these exposures may exist in the one case.

In such circumstances, the surgeon must operate with a light hand in order to avoid transgressing any of the slender barriers that remain intact. Granulations are not curetted. Fistulæ towards the labyrinth spaces are not probed.

Cholesteatoma is responsible for nearly 50 per cent. of the chronic suppurating ears that come to mastoid operation (J. S. Fraser), and the operator can recognize its presence by two characteristics: first, by the white cholesteatomatous lining membrane, and secondly, by the appearance of the bone.

The lining membrane of the cholesteatomatous cavity is often smooth and glistening, and devoid of granulations or other signs of the presence of bone sepsis. In such cases, Dundas Grant leaves the lining *in situ*, treating it as if it were a skin-graft of Nature's forming, and has recorded many successful cases treated in this manner. The writer removes it, fearing that it may harbour sepsis, and may conceal disintegrated bone.

The bone lining a cholesteatomatous cavity, when the membrane has been removed, is for the most part smooth, entire, and occupied by small shallow saucer- or basin-like hollows and depressions. But in certain regions, particularly in the antro-tympanic roof, the lining-membrane is frequently deficient, and then the bone will be found in a state of disintegration, breaking up under the pressure of the probe into small, crumbling gritty morsels, and revealing the dura mater. All such bone should be carefully removed.

(For the intracranial complications of Suppuration of the Middle Ear, see p. 531.)

Tuberculosis of the Ear is known to be a primary disease in infancy, affecting the tympanic mucosa in the first place, the infection, which is probably bovine and milk-borne (A. Logan Turner), passing up the relatively short and wide infantile Eustachian tube from the pharynx and naso-pharynx. In adults, it is occasionally met with as a secondary phenomenon in phthisis pulmonalis.

The tuberculous process in the ear consists of a slow, painless, progressive erosion and ulceration of the mucosa, the ossicles, and the bony walls, the end-products of which pass out into the external meatus and are avoided in discharge. The continual advance of the disease tends ultimately to the exposure and opening-up of the labyrinth spaces and meninges, with the production of labyrinthitis and meningitis, sometimes tuberculous in nature, but more often, perhaps, septic.

Symptoms.—In the smaller number of the infantile cases the onset is acute, with pain and bulging of the membrane, necessitating, it may be, paracentesis, but in most infants, as well as in older patients, the onset and progress of the disease are alike gradual and insidious, without any pain or discomfort apart from the discharge.

The discharge, when the infection is pure, is thin and curdy, but genuine pus is not uncommon as the raw ulcerating surfaces readily undergo secondary septic infection. Tubercle bacilli are visible in the discharge films when stained, but unless recourse is had to animal experiment, it is difficult to differentiate them from the other acid-fast bacilli common to all ear-discharges.

The tympanic membrane, on inspection, is seen to be pallid and may in early stages present multiple perforations, each one small and circular. Later on, these coalesce, and the membrane, together with the ossicles, undergoes entire removal by ulceration, the cavity left being lined with pale, flabby granulations, in typical cases.

The concurrence of insidious painless otorrhœa with enlarged peri-auricular glands in a child is strongly suggestive of tuberculosis, and the suspicion will be strengthened if facial paresis or paralysis is an early phenomenon in the course of the case, as tuberculosis in its progress quickly opens up the bony Fallopian canal and destroys the facial nerve.

The *diagnosis* of tuberculosis of the ear, however, is often difficult, as the features of the case may never present any difference from those of simple chronic suppuration.

Treatment.—In addition to the general treatment of tuberculosis by good food, tonics, fresh air, and cod liver oil, operation is often undertaken. The radical mastoid plan is followed, but the surgeon must endeavour to eradicate entirely the disease-bearing areas of bone, and this he may hope to do more successfully than in the ordinary mastoid operation, seeing that, if facial paralysis is present, he is no longer hampered by the presence of an intact facial nerve.

It is often difficult to remove all the disease and to cure the case. Nevertheless, operation is frequently successful in curing the disease.

CICATRICIAL CHANGES IN THE MIDDLE EAR—RESIDUAL SUPPURATION OF THE MIDDLE EAR (CICATRIZATION OF THE MIDDLE EAR, OTITIS MEDIA PURULENT RESIDUA, ADHESIVE PROCESSES IN THE MIDDLE EAR)

These various names are all given to the condition where as a result of the healing of previous suppuration, whether or not the disease has been accompanied with loss of substance, the parts are involved in granulation tissue which has undergone transformation into scar tissue.

Thus, the condition is, in brief, that of the involvement of the middle ear spaces and structures in scars.

A similar state of matters is found in certain cases of simple catarrhal otitis media where the secretion during the active stage may not have been anything more than mucous in character, and where the membrana tympani has never ruptured. And it is to this state that the name of Dry Catarrh, or Chronic Middle Ear Catarrh, is often given. As a matter of fact, the actual distinction between true active catarrh of the middle ear and the scar-tissue formation that results from it is difficult to make in any given case, and for that reason we shall consider that variety of scarring under the heading of catarrhs of the middle ear. (See p. 562.)

There is another reason for adopting this arrangement, and that is that the post-suppurative cicatrization is for the most part a non-progressive condition, whereas the catarrhal scar-formation is a progressive disease. Thus, the deafness after the middle ear has undergone cure from suppuration, may persist, but it usually persists without getting any worse, at all events, until late in life; while, on the other hand, the deafness resulting from catarrhal adhesions does become worse as time goes on, and may end in grave loss of hearing.

Pathology.—The extent of involvement of the middle ear in scar-formation depends naturally upon the type of the preceding suppuration. As might be expected, the cicatricial tissue is most abundant in and about the ossicles in the attic, and its subsequent contraction may induce great distortion, and a correspondingly great amount of obstructive deafness.

There is a popular idea that perforations of the membrana tympani never heal up. This is an error. Perforations, especially when they are small and central, but even when they are large and involve the margin of the membrane, often become entirely closed up by a cicatricial membrane, which is extremely thin, and which may be seen to flap in and out, when Siegle's speculum is used, independent of the rest of the tympanic membrane. In such cases, the amount of scar-tissue within the middle ear itself is often trifling; the new cicatricial membrane is not adherent to any part of the middle ear; and there is little or no interference with hearing. Naturally, in favourable cases such as these, the active disease has generally had but a short duration.

Not infrequently, however, we shall find, on examination with Siegle's speculum, that these cicatricial membranes are adherent to the inner wall of the tympanum at the promontory.

In cases in which the perforation remains patent, the persistence of the opening is due to the covering of its edges with epithelium from the outer aspect of the membrane.

The size of these dry perforations varies from an opening about 2 mm. in diameter to the absence of the whole vibrating membrane. In many of the cases it is possible to see through the perforation more or less of the chain of ossicles, and the promontory in the middle ear. The cicatricial contraction of the scar-tissue binds the ossicles together in distorted positions, the malleus handle being bent inward usually and adherent to the promontory, and as a consequence, the interference with function may be very considerable.

The hearing in these and similar cases, as a matter of fact, seems to depend chiefly upon the mobility of the foot-plate of the stapes in the oval window.

Scar-tissue, bands and adhesions binding down the rest of the ossicular chain, and pressing the stapes upon the oval window; and the presence within the niche of the oval window itself of dense scar-tissue immobilizing the foot-plate of the stapes—both of them produce serious deafness.

This scar-tissue may become calcified and ossified and lead to ankylosis of the stapedio-vestibular articulation, thus forming one variety of the so-called otosclerosis.

Symptoms.—Deafness of the obstructive variety is present, and it may be followed after a period measured by years by degenerative processes in the cochlea, giving rise to signs of nerve-deafness, sometimes of an extreme degree. Tinnitus may be experienced, but as a rule it is less pronounced than in the catarrhs and in true otosclerosis.

Vertigo is unusual even after the labyrinth is affected, and according to the writer's findings, the vestibular reactions are not interfered with unless the cochlea is involved, and then they become impaired.

The *Diagnosis* rests upon the discovery of scars, dry perforations, and deformities in the middle ear on inspection.

Prognosis.—As already said, deafness due to cicatricial changes in the ear tends to persist without much downward progress. In later life, the affection of the cochlea tends to severe deafness.

It is noticeable that if a patient deaf from cicatrices experiences a recurrence of discharge from the ear, his hearing is better during the attack. The reason for this obviously lies in the fact that the inflammatory reaction softens and loosens the cicatricial tissue binding down the ossicles.

It is necessary to remember that such recurrences of suppuration are by no means infrequent in these cases, and a characteristic and notable feature of such an event is the presence of pain for a prolonged period before the discharge appears. Probably this arises from the obstacle which the scar-tissue presents to small collections of pus finding an exit from small foci of bacterial irritation in the ear.

Perforations of the Membrane.

Treatment.—In recent and acute suppuration which is getting well, the tendency is for the perforation in the membrane to close spontaneously, but where this is delayed, the healing may be hastened by treatment. One of the simplest methods is to apply, after all discharge is quite stopped, a small piece of dry, glazed writing paper, cut a little larger than the perforation, in such a way that it covers the entire opening. The paper may be left, and by the time it falls off, the perforation will frequently be found to have closed.

Older perforations may also be closed by treating their edges with trichloracetic acid so as to raw them, and to encourage the growth of granulations, which may cover the opening.

There is a risk, however, in these and in all other active interferences, of inducing a re-infection of the middle ear, and a recurrence of suppuration. It is difficult to maintain asepsis in these recesses.

Efforts to remove the scar-tissue, or to loosen adherent ossicles by operation have frequently been made. But the difficulty of securing asepsis exposes the patient to the risks of further suppuration in the middle ear.

Artificial Membrane.—In a small proportion of cases with a dry perforation in the membrane, considerable improvement in the hearing can be obtained by applying a small disc-shaped pad of cotton-wool soaked in liquid paraffin over the perforation. This "artificial drum" should be applied by the surgeon himself first of all, and then the patient may be instructed how to insert it. A short thread should be attached to the disc in order to facilitate its removal, and it should be inserted in the morning and removed in the evening, a fresh one being made every day. Patients soon become expert in applying it to the proper place.

The "artificial drum" should not be used if the discharge is not quite at a standstill, and the patient should be watched for some days at its first insertions lest discharge should be induced by its presence.

For the treatment of the severer types of deafness, see p. 589.

INFECTIVE LABYRINTHITIS

(excluding Labyrinthitis from Cerebro-spinal Meningitis)

Anatomically, the labyrinth is an intracranial structure, but it is more convenient to consider its infective diseases at this place.

Four varieties of infective labyrinthitis will be described. First, serous labyrinthitis ; second, diffuse purulent labyrinthitis ; third, labyrinthitis associated with necrosis of the petrous bone ; and fourth, fistula of the labyrinth.

Serous Labyrinthitis in its pathology may be compared with the hypopyon that occurs in ulcer of the cornea. The labyrinth spaces are the seat of mild inflammatory changes as a result of toxic infection proceeding from septic infection of the middle ear spaces, without actual bacterial invasion. If the middle ear disease is cured or removed, the labyrinth irritation disappears, but if the middle ear infection leads to a bacterial invasion of the labyrinth then the serous labyrinthitis will become purulent.

Serous labyrinthitis seems to be a not uncommon complication of middle ear suppuration, either in the earliest stages of the infection or in the course of the chronic middle ear disease. Symptoms of serous labyrinthitis (labyrinth irritation) are fairly common also in the first few days following the radical mastoid operation, especially if there has been a free curetting of granulations on the labyrinth wall of the antro-tympanic cavities.

Symptoms.—The onset is usually sudden, and the symptoms partake of the nature of the "labyrinth storm" (see p. 417) of more or less severity. As a rule, the patient is unable to get about, and sickness is experienced, but sometimes the symptoms of the attack are quite mild.

If seen early in the attack spontaneous nystagmus of a pronounced character is manifest, but in typical cases the symptoms subside in two or three days, and the intensity of the nystagmus is reduced correspondingly.

Serous labyrinthitis is said to leave no permanent ill-effects, and in most cases this is true. Nevertheless, in several observed by the writer after the mastoid operation, a certain amount of nerve deafness followed, and, at the same time, the vestibular reactions became decidedly impaired.

The occurrence of attacks such as these during the course of a chronic suppuration of the middle ear constitutes an indication for the radical mastoid, if that operation has not already been performed, but it should be suspended until the intensity of the symptoms has subsided.

Diagnosis.—The symptoms are sufficiently clear to indicate labyrinthitis, but it is always difficult, and often it is impossible to tell when the labyrinthitis is serous and negligible, and when it is purulent and dangerous.

The knowledge of an injury to the membranous labyrinth, as during a mastoid operation; the presence of pain in the ear; of sudden and severe nerve deafness; of occipital headache; of cervical rigidity;—any of these would suggest the purulent form of the disease.

Lumbar puncture ought to be performed as a routine in acute labyrinthitis, and indeed, as Archer Ryland has pointed out, it is a safe precaution to adopt it in all cases of ordinary acute mastoiditis. (See p. 557 for Lumbar Puncture.)

Treatment.—The patient is kept in bed, and, in undoubted cases of serous labyrinthitis, no further treatment is required.

In any event, two or three days may elapse while watching the symptoms closely, before deciding upon any active treatment.

But after the acute phenomena have subsided, then, if the radical mastoid operation has not been performed, it ought to be, without delay. On the other hand, if that operation has already been performed, no further interference may be required —unless the symptoms recur again at a later date.

Purulent Labyrinthitis.—In this variety of the disease the membranous labyrinth, being invaded by septic organisms, becomes the seat of suppuration. The invasion takes place through a breach in the outer labyrinth wall, usually in the neighbourhood of the round or oval windows, but the infection may also reach the spaces through a fistula in the semicircular canals, an opening which is the product, as a rule, of cholesteatomatous disease. Purulent labyrinthitis may also be induced by infection of a labyrinth which has been exposed to tuberculosis. Finally, a breach of the wall may be produced by traumatism in operating, as by dislocation of the stapes, an accident which, unless speedy precautions are taken, is apt to be followed by a general infection of the labyrinth.

In the more chronic forms of destructive middle ear disease, such as cholesteatoma, the advance of the disease-process into the labyrinth is so slowly effected, that there is time for barriers to the general infection of the labyrinth to be erected, and the disease-areas are shut off by fibrous tissue and even by bone. Indeed, the operation of the protective agencies in the labyrinth is not limited to cholesteatomatous disease. Even in more rapidly advancing lesions, efforts at repelling or confining the disease are common, in the form of granulation-tissue formation.

The danger of infective labyrinthitis lies in the fact that the spaces of the membranous labyrinth lead directly to the meninges, and that intracranial complications such as purulent meningitis or cerebellar abscess are almost inevitable if the advancing disease cannot be stayed.

Symptoms.—The symptoms of purulent labyrinthitis are at the outset in most cases indistinguishable from those of serous labyrinthitis, but in the purulent disease, as time goes on, although the phenomena of the labyrinth storm pass away, the internal ear does not regain its function. Severe nerve deafness persists and the vestibular tests will show a continued absence of the vestibular reactions.

In many cases, the symptoms merge into those of the intracranial complication set up by the labyrinth infection. It is thus a frequent experience to find that the spontaneous nystagmus of the labyrinth invasion, instead of becoming less in a few days and passing gradually away, as is the rule in uncomplicated labyrinthitis, retains its severity unimpaired, while the subjective giddiness and the Rombergism also continue.

Such continuance arouses the suspicion of cerebellar abscess.

Or, pain in the occipital region accompanied with some cervical rigidity sets in and raises the question of meningitis.

Here, as in the last condition, lumbar puncture should be performed and the cerebro-spinal fluid examined without delay, and the examination should be repeated every day if the symptoms continue, while a close daily watch should be maintained in order to obtain the first warnings of any intracranial extension.

The *pyrexia* in purulent labyrinthitis seldom exceeds 101° F., and after the acute stage, although the suppuration is still present, it may sink to and remain about normal. In this respect, it differs little, if at all, from the serous type of the disease.

Spontaneous Nystagmus.—No reliance can be placed upon the *direction* of the nystagmus as an indication of the particular ear affected. Sometimes, it is directed to the affected side; sometimes it is directed to the sound side; or it is directed first to one side, and then to the other. The presence of spontaneous nystagmus, however, is a sign of involvement of the vestibular tract, and should be regarded with misgiving.

As long as the spontaneous nystagmus continues, it is impossible to make use of the vestibular tests. But after the vertigo and spontaneous nystagmus have subsided, their employment will, in purulent labyrinthitis, as we have already said, show the vestibular sense to be in abeyance.

The *hearing*, on the other hand, may be tested at any period. In purulent labyrinthitis, there is serious, and often absolute nerve deafness upon the affected side. In serous labyrinthitis, a certain amount of hearing is retained even during the active stage of the disease. But the fact remains that for the first few days it is generally impossible to say whether the patient is suffering from serous or from purulent labyrinthitis.

Prognosis.—The prognosis of purulent labyrinthitis, as regards life, is grave, until, at least, the labyrinth has been opened and drained, an operation which has proved itself, in skilled hands, to be life-saving.

As regards function, the prognosis is, of course, bad. A modicum of hearing may remain, but in most cases it is entirely destroyed. The same is true of the vestibular organ, but its loss is subjectively imperceptible, or nearly so.

Treatment of Purulent Labyrinthitis.—We are compelled to temporize until the nature of the disease declares itself, because, while in the purulent variety delay is dangerous, on the other hand, to perform the labyrinth operation upon a simple serous labyrinthitis exposes the patient not only, perhaps, to a real infection of the labyrinth, but also to complete or almost complete deafness.

Another feature of this condition which is a further source of embarrassment to the surgeon is, that, as nearly all otologists agree, if a patient with latent purulent labyrinthitis has the radical mastoid operation performed without the labyrinth being opened, he runs a very grave risk of meningitis supervening two or three days after the operation.

It must be confessed, therefore, as things are at present, that the indications in any given case are often very obscure.

Many surgeons withhold decision until they perform the radical mastoid operation, and are guided by what that operation discloses. Here, again, however, it must be admitted that no very definite lead one way or another may be obtained.

One point is clear, and that is not to perform any operation during the first two or three days of the acute labyrinth phenomena.

On the other hand, if the diagnosis is undoubted, and especially if intracranial phenomena (see p. 531) are beginning to manifest themselves, then the labyrinth must be opened and drained without delay.

LABYRINTHOTOMY (Double Vestibulotomy)

Description.—The opening and draining of the membranous labyrinth through its outer wall.

Indications.—The signs of purulent labyrinthitis especially when accompanied by symptoms of intracranial invasion, such as persistence of spontaneous nystagmus, cervical rigidity, and leukocytosis of the cerebro-spinal fluid.

(Apart altogether from infective labyrinthitis, the labyrinth has been opened or removed for the cure of inveterate vertigo, and of intractable tinnitus.)

Contra-Indications.—It is generally considered unsafe to proceed to the labyrinth operation until two or three days have elapsed from the onset of acute labyrinth symptoms, the reason being, first, that the symptoms may be due to simple serous labyrinthitis, and secondly, that if infective, the lapse of a few days may enable the natural defences to be organized.

Operation.—The radical mastoid operation having been performed, the bony posterior meatal wall is chiselled down as flat as possible, the inner antro-tympanic wall is assiduously cleared of all granulations, and the patient's head is turned with the face half upwards, so as to bring the neighbourhood of the oval window as well as possible within view. With the fine labyrinth probe, search is made in the inner antro-tympanic wall for a fistula, and if any is found, it is made the site of the operative opening into the labyrinth. If none is found, two points of election exist for the artificial openings; the first, behind the Fallopian canal and the facial nerve, into the external semicircular canal in the inner wall or floor of the aditus. It is known by a smooth, oval bulge or prominence in this position.

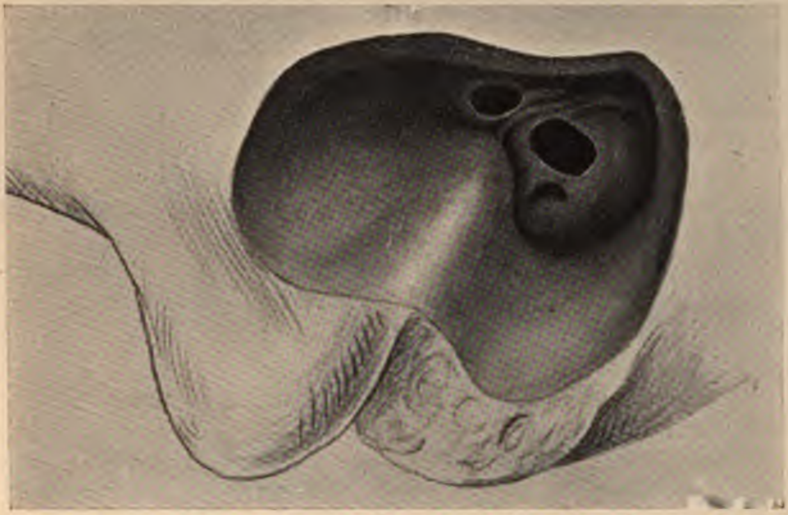

FIG. 188.—The radical mastoid bone cavity (diagrammatic), showing the openings made through the inner (medial) tympanic wall in double vestibulotomy (Labyrinthotomy). Other features as in Fig. 184.

The opening is made through the hard bone of the petrous into the canal with a fine labyrinth gouge and mallet. Once the canal is reached and opened, the opening may be extended, under the guidance of the probe, as far backward as the operator desires.

Turning now to the tympanic region, the foramen ovale is enlarged with the fine gouge in a forward and downward direction, and through the opening thus made, the interior of the labyrinth spaces may be curetted if required. (Fig. 188.)

The probe entered at either of these artificial openings, and directed towards the other, will pass behind the bridge of bone in which is lodged the Fallopian canal and the facial nerve (Sir William Milligan).

B.I.P.P. is applied to all the raw surfaces, and the post-aural incision is closed. As drainage is all-important, no graft is applied, neither is the wound packed with gauze. But if there is a free flow of cerebro-spinal fluid sufficient dressing should be applied to absorb it.

Difficulties and Dangers.—When the parts are well opened up and anatomically well displayed, labyrinthotomy is an easy operation, and need not occupy more than a few minutes. But if the antro-tympanic cavities are small, deep, and highly situated, it is extremely difficult to locate the various structures and to direct the gouge accurately. It should be remembered that the parts here are really minute, the distance from the oval window to the modiolus and the internal auditory meatus is not any more than two millimetres. Thus, the modiolus may easily be wounded, and the internal auditory meatus opened up, a free flow of cerebro-spinal fluid resulting. With good drainage, however, this accident does no harm.

The facial nerve is liable to be wounded or divided in the bone operation. But the accident need not happen if due care and precision in operating be secured.

After-Treatment.—The patient should be watched for intracranial complications, for meningitis and cerebellar abscess particularly, and kept in bed for a fortnight at least after the operation.

Syringing of the wound or of the interior of the ear is not recommended.

Results.—In uncomplicated purulent labyrinthitis, the results are good, healing taking place quite rapidly. Indeed, it is often noticeable that if labyrinthotomy has been performed upon an old radical mastoid cavity which has been obstinately discharging the result of the deeper operation often is to bring about a rapid drying-up and epithelialization of the mastoid cavity. The reason obviously is that the labyrinth operation removes the last remnant of active bone disease.

(It should be stated that Neumann's method of opening the labyrinth differs from the above. He removes the posterior surface of the petrous bone until the posterior and horizontal semicircular canals are cut across; then he advances as far as the base of the modiolus, opens up this end of the acoustic nerve, and finally removes the promontory.)

Necrosis of the Petrous Bone involving the labyrinth.—Necrosis of more or less of the petrous bone with part or whole of the canalicular and cochlear regions is produced when uppuration of a virulent character attacks a temporal bone n which the petrous process contains air-cells communicating with the mastoid cells or tympanic cavity, and surrounding the hard dense bone of the cochlear and canalicular regions (the labyrinth capsule). These peri-labyrinth cells become filled with pus and granulations, and the blood-supply to the interior being thereby cut off to a greater or less extent, the bone of the labyrinth undergoes necrosis.

Obviously, this represents a condition of gravity, but it is remarkable how many such cases recover without any permanent ill-effect beyond loss of hearing and perhaps persistent facial paralysis.

Symptoms.—The attack on the peri-labyrinth bone is attended with the signs of the labyrinth storm and with severe nerve-deafness if the cochlea is involved. As in other acute labyrinth diseases, those preliminary symptoms soon subside, and the case passes into the category of chronic suppuration of the middle ear, in which, however, we discover severe deafness when the cochlea is implicated; absence of the vestibular reactions when the canals are affected; and facial paralysis when the aqueductus Fallopii is involved in the necrotic process.

The sequestrum, in course of time, undergoes exfoliation, and may be disintegrated and voided in the ear discharge, or it may be removed *en masse* from the meatus. If the case comes to operation, the sequestrum will be found occupying the region of the labyrinth, and will be removed by the surgeon.

Diagnosis.—The symptoms are those of purulent labyrinthitis, but if facial paralysis is present, necrosis may be looked for, as purulent labyrinthitis seldom produces facial paralysis.

This remark does not apply to facial *paresis*, since a paretic condition of the face is a common event in basal meningitis secondary to purulent labyrinthitis, and is much less common than paralysis in petrous necrosis (1 : 50).

In the absence of facial paralysis, the discovery of the sequestrum embedded in granulations is usually made accidentally in the course of the radical mastoid or labyrinthotomy operation.

Prognosis. — Bezold's estimated mortality of labyrinth necrosis amounted to 20 per cent. only. But it must be remembered that these figures must necessarily ignore cases which have been fatal at an early stage in consequence of a failure of the organism to limit the disease in the petrous.

For cases in which the sequestrum has been discovered, and the condition diagnosed, Bezold's figures may perhaps be taken as approximately accurate.

Treatment.—The presence of ear suppuration combined with severe nerve deafness, or with absence of the vestibular reactions; or with facial paralysis; or with any combination of these three conditions would lead to an immediate radical mastoid operation, and perhaps labyrinthotomy. And in the course of the operation, a sequestrum, if present, would be found and removed.

In removing the sequestrum, gentleness is to be exercised, as the facial nerve may be torn through; and indeed even more serious consequences may follow forcible removal, such as opening up and exposure of the meningeal spaces to infection, or rupture of the wall of the jugular bulb, or conceivably, indeed, of the internal carotid artery.

Fistula of the Labyrinth.—Fistula of the outer wall of the labyrinth is invariably the result of cholesteatomatous disease, and its usual site is on the prominence of the external semicircular canal in the aditus, where it can be seen when the parts have been opened up in the radical mastoid operation as a small oval area with the long diameter horizontal, occupied by a dark-red granulation. Fistula may also form in the tympanic wall of the labyrinth about the oval window, but it is probably less common there.

In most cases it represents merely a local invasion, the rest of the labyrinth being unaffected—being, indeed, shut off by the granulation tissue in and about the fistula.

This favourable state of matters, however, is not invariable. The labyrinth may be the seat of extensive disease with its spaces full of pus and granulations, and its functions in total abeyance.

Symptoms.—The symptoms obviously depend upon the condition of the labyrinth. When the fistula exists with an active labyrinth, hearing and the vestibular tests will be little, if at all, affected, but there is present a sign of considerable value in the "fistula symptom." (See p. 419.)

This sign in a patient with cholesteatoma, and a history of occasional vertigo of a transitory nature, enables a diagnosis of fistula to be made with fair confidence.

But, the same sign exists in early deafness from congenital syphilis, for some unknown reason, and in excessive mobility of the stapes (Politzer).

Prognosis.—Unless operated on, fistula in the labyrinth may prove at any moment to be the starting point of a generalized labyrinthitis with all its attendant risks, but if operation is performed, the prognosis is good, at all events, as regards labyrinth infection.

Treatment.—Cases of fistula in the labyrinth should be operated on without any delay ; and the radical mastoid operation is generally sufficient. If the labyrinth is active, the granulation in the fistula should be left alone, as curetting of the fistula is apt to lead to generalized labyrinth infection. If, however, the labyrinth is inert—grave nerve deafness being present with an absence of vestibular reactions—the labyrinth should be opened and drained.

A fistula in the labyrinth contra-indicates grafting the radical mastoid wound.

Result.—The simple radical mastoid, if thoroughly performed, is usually sufficient to lead to closure of the fistulous opening by scar-tissue, and in many, perhaps in most cases the physiological activity of the labyrinth is left unimpaired. It happens occasionally, however, as we have already seen, that the mastoid operation is followed by an attack of serous labyrinthitis, which although itself but transitory, leads to considerable reduction in the hearing power and in the vestibular activity.

INTRACRANIAL COMPLICATIONS OF OTITIC ORIGIN

Extension of the infective process to intracranial structures from a suppurating middle ear constitutes the ever-present peril that haunts every case of middle ear suppuration.

These complications will be dealt with in the following order :

1. Extradural abscess.
2. Thrombosis of the lateral sinus.
3. Cerebellar abscess.
4. Cerebral (temporo-sphenoidal) abscess.
5. Septic meningitis :
    - (*a*) purulent ;
    - (*b*) serous.

EXTRADURAL ABSCESS

Extradural abscess is an abscess lying between the bony cranium and the dura mater ; it is, therefore, not in direct contact with the brain or soft membranes, and for that reason it is less dangerous to life than the other intracranial complications. While this is true, however, we must remember also that if an extradural abscess is not promptly evacuated, it may ulcerate its way through the dura mater, and lead to a fatal infection of the lateral sinus, the soft membranes, or the brain.

The most usual *site* of extradural abscess is in the lateral sinus groove, the infection reaching this locality by way of the antrum or the mastoid cells, and constituting one of the commoner complications of mastoiditis. It is not infrequently the result of a necrotic process of the bone of the sinus groove.

In this situation it is liable to lead on to lateral sinus thrombosis, but in most of the cases, although the wall is covered with granulations, the interior of the sinus remains healthy, as the uneventful course of most cases, after the abscess has been evacuated, clearly proves.

Pus is confined to the sinus groove by the attachment of the dura mater to the bony edges of the groove, and it may be seen to be under considerable pressure when first opened into with the chisel. It occasionally finds its way from the groove through the foramen for the mastoid emissary vein on to the outer surface of the skull.

Extradural abscess also occurs on the intracranial side of the roof of the tympanic and antral cavities, and may attain considerable dimensions and extension, but it is not common here as this bony roof, being thin, undergoes early destruction or absorption so that a retention of pus in this situation does not readily occur.

In the rare otogenic osteomyelitis—a disease of infancy and childhood—extradural abscess may be encountered; and, finally, under the name of *saccus empyema*, attention has been drawn to a small abscess formation on the posterior surface of the petrous bone, secondary to purulent labyrinthitis and reaching this region by way of the aquæductus vestibuli and the saccus endolymphaticus. The importance of this abscess lies in the fact that it is a frequent forerunner of cerebellar abscess or of meningitis, being one of the stepping-stones from the labyrinth to the brain. (Fig. 142.)

Symptoms.—In the great majority of cases extradural abscess is latent, its presence being accidentally discovered at operation. Very rarely, indeed, does one venture to diagnose its existence from symptoms. But it may be suspected and looked for when there is severe and precisely circumscribed headache with tenderness on percussion of the skull in the neighbourhood. Another highly suggestive combination of events consists in the occurrence of severe headache with mental dullness, and perhaps signs of raised intracranial pressure, which is paroxysmal in character, the relief of the symptoms coinciding with the appearance of a free flow of pus from the ear through the external auditory meatus. Such a conjunction of phenomena, especially if recurrent, would indicate a leaking extradural abscess as the most likely explanation. But it is not the only explanation.

The appearance during a mastoid operation of pus under pressure welling out from a small orifice in the neighbourhood of the lateral sinus groove or of the antro-tympanic roof is usually the first indication we receive of extradural abscess. This appearance should, of course, lead to a removal of bone, and an exposure of the source of the purulent stream.

Prognosis.—The prognosis of extradural abscess is good when once it has been evacuated and its cavity freely laid open. But, as it may be associated with lateral sinus thrombosis, and with abscess in the brain, the case should be closely watched for symptoms of these diseases during convalescence.

Treatment.—A mastoid operation is performed, and the bone of the inner cortex of the skull is removed so as to expose the dura and to empty the abscess cavity. The removal of bone should be freely effected in order to obviate the re-accumulation of the pus. After evacuation, the surface of the dura, which is covered with granulations, is carefully and gently probed for a fistulous opening or tract which may lead the surgeon to an abscess in the brain. But the granulations should not be curetted.

SEPTIC THROMBO-PHLEBITIS OF THE LATERAL SINUS (LATERAL SINUS THROMBOSIS)

The lateral (sigmoid) sinus deeply grooves the inner aspect of the bony cranium in its course from the torcular Herophyli to the bulb of origin of the internal jugular vein—the jugular bulb (Fig. 189)—and this course brings the sinus into close relationship with the bone on the inner aspect of the mastoid process, where it is necessarily exposed to infection during septic mastoiditis when (*a*) the bone contiguous to the sinus is diseased, or (*b*) pus from the mastoid cells finds its way into the lateral sinus groove (*perisinous abscess*—see the extradural abscess,

p. 531). This latter mode of production is not common as it happens.

Secondly, the veins of the mastoid process, and of the antro-tympanic cavities, drain into the lateral sinus, and, in purulent otitis and mastoiditis, may themselves become the seat of a septic phlebitis which extends along these channels to infect the interior of the lateral sinus independent of any bone disease.

Finally, as venules occasionally pass from the middle ear through the bony floor of the tympanic cavity to the jugular bulb below, it sometimes happens that the jugular bulb becomes infected in simple purulent otitis media without either mastoid disease being present, or thrombosis of the lateral sinus proper.

An occasional cause of lateral sinus thrombosis is the accidental operative exposure and injury of the dural wall of the healthy sinus during the performance of the radical mastoid operation, the symptoms of the complication appearing about ten days after operation (Hunter Tod). This unwelcome sequence is unlikely to occur if the exposed sinus wall is well smeared over with B.I.P.P. at the mastoid operation.

Sinus phlebitis is a septic inflammation of the vessel wall, upon the inner side of which the circulating blood adheres and coagulates, the coagulum in its turn becoming infected and breaking down into a purulent or semi-purulent and disintegrated mass. The disease has a tendency to spread far beyond the site of origin by the successive and continued formation of coagulum in the vessel lumen.

Thus, the process readily spreads from the lateral sinus in the direction of the blood-current to the jugular bulb and down the internal jugular vein in the neck, where, indeed, it may reach as far as the subclavian vein. It may also extend upwards to the torcular, and pass into the lateral sinus of the opposite side. Lastly, by way of the superior or inferior petrosal sinuses, it may reach the cavernous sinus, setting up cavernous sinus thrombosis.

The *Symptoms* are those of acute septicæmia, and occasionally of pyæmia, and as the constitutional phenomena predominate, the local disease is very apt to be overlooked, especially as there may be no sign or symptom of any aural trouble beyond the otorrhœa. Pain, headache, tenderness and swelling over the mastoid are as often as not entirely absent, and the patient mentally is quite clear. During the first week or ten days of his illness, he may, indeed, be able to walk about, and to attend to his ordinary affairs.

But the constitutional phenomena are nevertheless very striking, and sooner or later obtrude themselves even upon a careless or inattentive observer. The symptoms which first attract attention are, in these ambulant cases, increasing asthenia and rigors.

The rigors form part and parcel of the septicæmic group of phenomena, and occur during the upward rise of a remitting or intermitting pyrexia, the oscillations of which are both rapid and extensive. Thus, the temperature may swing from 99° F., or even from the normal line to 104° or 105° F. not only once, but even sometimes twice, or oftener, in the twenty four hours; and, in typical cases, as we have already said, each time the rise occurs, it is marked by a definite and sustained rigor, followed by profuse perspiration.

But it should be noted, however, that the rigors may be so slight and inconspicuous as to escape notice. Indeed, if the patient is kept in bed, they may be entirely absent, and in children also they generally are absent. So that their absence must never be regarded as a sign that lateral sinus thrombosis is not present.

With those evidences of profound toxic absorption, there is usually also headache, which, as it is most commonly frontal, is also, no doubt, toxic in origin. And as time goes on, the patient's weakness goes on increasing, and emaciation becomes extreme.

In uncomplicated cases, the mentality remains clear until the end, and optic neuritis and other signs of raised intracranial pressure are absent.

A text-book symptom-series, which has obtained a wide popularity, is that of tenderness, swelling, and a feeling of a "cord" in the course of the internal jugular vein in the neck.

Those signs are rare, and they will become still more rare in accordance with the early surgical intervention of modern operators. Operation should never be deferred pending their appearance.

Examination of the ear reveals nothing but the usual signs of purulent otitis media. In simple lateral sinus thrombosis, there is no mastoid pain or tenderness, and no mastoid swelling proper. There is, however, and the writer wishes to emphasize this sign, in most cases even at an early stage œdema of the scalp towards the occipital region posterior to the mastoid process, and the method to be employed in eliciting this sign is that of continued hard pressure with one finger as was described in discussing mastoiditis. (See p. 495.)

As the case progresses, the pulse becomes rapid and weak; some jaundice may be present; the tongue is coated and dry, and the spleen becomes enlarged.

Course and Termination.—The duration of the illness, independent of surgical intervention, is, like that of all other "blood-poisonings," very variable. A minority of cases die within a day or two from overwhelming toxæmia, or from the formation of metastases in vital organs. A few linger on for weeks or even months with a succession of pyæmic abscesses. The majority of cases last from a month to six weeks, and die as a result of septic meningitis, septic broncho-pneumonia, or general toxæmia. In some, the end is hastened by the extension of the thrombosing disease to the cavernous and other sinuses of the cranium.

A few cases of spontaneous recovery are on record, so that this termination is not unknown. But such a fortunate termination cannot be looked for, since practically all cases die if not relieved of the disease by operation.

Diagnosis.—Lateral sinus thrombosis has been, and is still, frequently overlooked, under the diagnosis of broncho-pneumonia, or meningitis.

On the other hand, malaria occurring during an acute otitis media or mastoiditis is apt to be mistaken for sinus thrombosis (Neil Maclay).

The remitting temperature and septic diarrhœa may lead to the fallacious diagnosis of typhoid fever, but the Widal reaction is negative in lateral sinus thrombosis, and regularly recurring rigors, when these are present, should be sufficient to turn the mind to the search for a septic focus.

Signs of lateral sinus thrombosis may occur without any thrombus being found in the vein when it is opened by operation. These are cases of septic infection from the ear, the so-called *bacteræmia*. In cases without rigors it may be impossible to avoid such a mistake being made. Fortunately, as the sequel shows, the error is unimportant, as the opening of a healthy sinus does not seem to do any harm, and the operation is usually followed by a fall in the temperature to normal.

Prognosis.—Unoperated cases are all, or nearly all, fatal. But the early and complete opening up of the thrombosed sinus is followed by rapid recovery in a majority of cases. The longer operation is delayed, the less likely is recovery to take place.

The occurrence of pyæmic abscesses indicates a prolonged illness which may prove fatal by exhaustion. But severe septicæmia is more to be dreaded than is pyæmia with septic metastases in the subcutaneous tissue or joints.

Cavernous sinus thrombosis is very seldom recovered from. (See p. 542.) And the presence or onset of other intracranial complications is of grave significance. Of these, meningitis and cerebellar abscess are the most common accompaniments or sequelæ of lateral sinus thrombosis, and the case should be examined daily for the early signs of their presence.

Lateral sinus thrombosis occurring in chronic suppuration of the ear is said to be more dangerous than when it appears in the acute disease.

Treatment.—The focus of septic infection in the lateral sinus must be removed, and this can only be done by exposing the vessel, opening it up, and clearing out the infected clot. As an additional measure of precaution, the internal jugular vein in the neck is exposed, ligated, and a portion of its length resected. When the thrombotic process has extended into the jugular vein, this step in the operation is imperative, and the removal of the vein must be carried down to a point below the termination of the clot if that is possible. When, however, the thrombosis is confined to that part of the lateral sinus that can be completely cleared out through an opening in the skull, the indication for resecting the jugular vein in the neck is not quite so obvious, and a considerable body of expert opinion favours a limitation of the operation, under those circumstances, to the lateral sinus alone. The resection of the jugular vein in all cases, however, is advisable, chiefly because it is difficult to be sure that all the disease has been eradicated in any given case, but also because this detail adds but little to the severity of the operation, and, as far as I am aware, it is not followed by any ill-effects. Severing the jugular vein does not, it is true, cut the septic focus entirely out of the circulation, but it shuts up the only short and direct route. Operators may regret not having resected the jugular vein ; they will never regret having done so.

The ligation and resection of the jugular vein should be performed at the same *séance* as the opening of the lateral sinus, and some operators wisely proceed to the neck operation as soon as they have ascertained that the lateral sinus contains clot, and before clearing the clot out of the sinus.

The diagnosis of lateral sinus thrombosis cannot always be arrived at in its early stages, and yet it is during this period that operation is most successful. But the operative exposure and examination of the interior of the lateral sinus is free from danger, consequently we are justified in operating on suspicion. In other words, a temperature oscillating as we have described associated with discharge from the ear should lead to an exploration of the lateral sinus through the mastoid process not later than three days after the onset of the pyrexia—earlier if the signs are more pronounced; if, that is to say, there are rigors and signs of mastoid, or, *à fortiori*, of intracranial mischief.

In cases with discharge from both ears, it may be impossible to determine which side it is that is infected. In such cases, our only guide is the local condition, and we operate upon the side which is painful, or which shows more inflammation. An unfortunate decision may entail a fatality.

OPERATION ON THE LATERAL SINUS

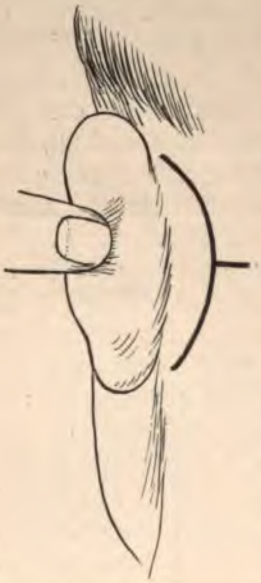

FIG. 190.—Post-aural incisions for cortical mastoid and lateral sinus or cerebellum operations.

Operation.—The mastoid process having been opened, and either the cortical or the radical operation having been performed, a horizontal incision from one to two inches in length is made from the centre of the post-aural incision backwards through the tissues of the scalp, and carried down to the bone of the cranium. (See Fig. 190.) The flaps of soft tissue thus formed are raised, and retracted so as to expose freely the bone of the posterior mastoid and occiput. With chisel and gouge, the cavity in the mastoid bone is enlarged backwards by a removal of the bone until the lateral sinus groove is opened sufficiently to admit the blade of cutting bone forceps. With these forceps, the bone covering the lateral sinus is now removed in a direction upwards and backwards towards the torcular, and downwards and inwards in the direction of the horizontal limb of the sinus. When about an inch and a half of the sinus wall has thus been laid bare, it is inspected for discoloured areas or areas of disease. It may also be palpated for underlying clot, and the presence or absence of intercranial pulsation observed.

The identification of the sinus is not always easy. The healthy sinus wall is smooth and pale blue. But if the vessel wall and the adjoining dura are covered with granulations, which here are tough and fibrous, it may be, indeed, very difficult to distinguish the one from the other.

Having identified the sinus, a long strip of gauze is taken and laid conveniently near the field of operation; then by means of a sharp scalpel, the vessel is opened, and its wall slit freely up longitudinally for about an inch, the incision being made to traverse any obviously diseased patches.

If the sinus is occluded by clot, this incision is not followed by any bleeding.

If the vessel is not occluded, however, immediate and very free hæmorrhage does take place. But, seizing the gauze-strip before mentioned, the surgeon quickly packs it into the sinus groove and under the bone at the upper and posterior part of the groove in such a way that it compresses the sinus above the slit made in its walls. If, at the same time, retrograde bleeding from the jugular bulb is taking place, a plug must also be inserted in the lower part of the bony opening of the sinus groove.

The hæmorrhage having been stopped by these measures, we seize the vessel wall with forceps, everting the edge of the incision, and proceed to examine the interior of the vessel. If healthy, it is smooth and white. A coagulum is also looked for, and this, if recent, is reddish; if old, grey or whitish. If no coagulum is found, the lower plug is now removed, and a fine curette passed along the interior of the vessel towards the bulb, and withdrawn so as to hook out any clot that may be lying in that direction.

If none is found, the exploration has been negative, and the packing in the sinus groove having been replaced with iodoform gauze, the wound is dressed open, and the packing is left *in situ*, and undisturbed for three days. On its removal then, bleeding seldom occurs. If it does, a packing is re-inserted.

This is the only satisfactory method of exploring the sinus. Other plans such as the use of a hypodermic or other hollow needle may traverse a mural clot, and give no indication of its presence.

When a coagulum is found, many operators make a practice of proceeding at once to the jugular resection (see below). In any case, the clot in the lateral sinus is spooned out of the vessel by means of a curette, the removal of the coagulum towards the torcular being undertaken first. The clot is removed until free bleeding from above shows the vessel to be clear. Then the sinus groove having been packed as described above, the vessel is cleared out in the direction of the jugular bulb. Finally, B.I.P.P. is applied, and a loose packing inserted into the wound.

RESECTION OF THE JUGULAR VEIN

A freshly sterilized set of instruments is used.

An incision is made through the skin of the neck, the platysma, and superficial layer of the deep cervical fascia along the anterior border of the sterno-mastoid muscle from the level of the angle of the jaw to the level of the lower border of the thyroid cartilage —or further if necessary. The anterior border of the sterno-mastoid having been defined and cleared, the opening is uniformly deepened by blunt, and occasional sharp dissection until the jugular vein is exposed, the guide to which is the deep facial vein crossing the field of operation in its upper part above the level of the corner of the hyoid bone. The jugular vein seems to lie well behind as well as deep to the anterior border of the sterno-mastoid muscle. The vessel having been exposed and cleared along the full extent of the wound in the neck, the facial vein is also cleared and divided between two silk ligatures. Then a pair of ligatures are applied to the jugular vein at its upper end, and another pair at the lower end, and the vein is divided at either end between those ligatures. (Fig. 191.) Having thus resected as much of the vein as is required, the vessel is opened and inspected for coagulum. If it contains clot, and the lower end of it has not been reached, the wound must be extended downwards as far as is necessary to obtain the vein free from coagulum. This may imply the further ligature of the thyroid veins, superior and inferior, entering the internal jugular.

The wound is now sutured in layers with catgut, and the skin incision closed, but the ligatured upper end of the divided jugular vein is brought out of the wound on to the skin surface, as, since it is presumably highly septic, it may contaminate the neck wound.

As in all neck dissections, each bleeding-point should be ligatured before the wound is closed, and a drainage tube should be inserted for twenty-four hours.

After-treatment.—Dry dressing is applied for twenty-four hours, and is then followed by fomentations—to the mastoid wound, at all events, as the sepsis is often virulent. The gauze packing is removed daily unless there has been sinus hæmorrhage.

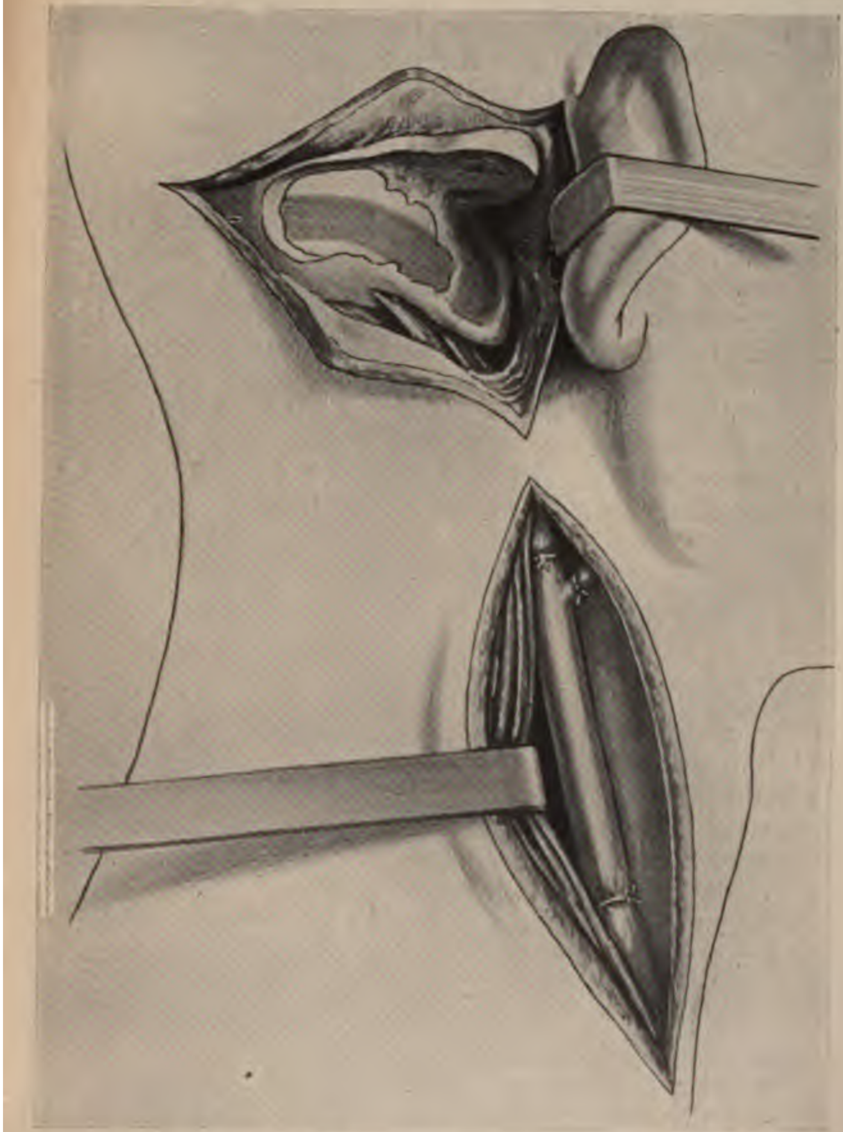

FIG. 191.—Exposure of the Lateral Sinus and Jugular Vein in the Neck. The latter is ligatured above and below. The facial vein is also ligatured, and the sterno-mastoid is retracted (after Laurens).

A careful outlook is maintained for signs of meningitis and the cerebellar abscess.

Septic Thrombo-phlebitis of the Cavernous Sinus is an uncommon sequel of lateral sinus thrombosis, and, indeed, it is more frequently due to nasal, pharyngeal, or facial infections than to aural infections.

Symptoms.—The characteristic feature of cavernous sinus thrombosis is the protrusion of the eyeball of the affected side with chemosis, and with redness and swelling of the eyelids followed in a few days by similar appearances in the other eye. The fundi show choked discs; vision is interfered with, and in the later stages there is ophthalmoplegia. At the same time the constitutional symptoms of septic venous thrombosis are present.

The disease is almost invariably fatal, although a few recoveries have been recorded; one by the author.

Treatment.—No method of opening and draining the cavernous sinus has so far been devised and successfully carried out.

CEREBELLAR ABSCESS

The cerebellum is exposed to infection when pyogenic micro-organisms reach the dura of the posterior fossa in (*a*) lateral sinus thrombosis; (*b*) mastoid suppuration, especially when the bone of the posterior aspect of the petrous deep to the lateral sinus groove is involved; (*c*) purulent labyrinthitis producing saccus empyema. (See p. 532.)

It is not always possible to discover the route by which the infection reaches the cerebellum, but sometimes the route is quite obvious as when the dura shows areas of granulations, and even, it may be, a fistula leading into the brain substance.

Cerebellar abscesses are generally small and superficial, involving the brain substance contiguous to the mastoid and petrous. But there are exceptions to this, and cerebellar abscess may be found deep in the cerebellum, even occupying a position posterior to the lateral sinus.

The *Symptoms* are those of brain abscess coupled with certain localizing signs which can generally be found if sought for.

Abscess in the brain, uncomplicated with meningitis or lateral sinus thrombosis, after an initial stage during which the temperature for a few days may be slightly elevated, induces headache, persistent and severe, with occasional bouts of vomiting, with a slow pulse (50 to 60 in the adult lying down) and a subnormal temperature.

These symptoms, which are due to raised intracranial pressure, are common to all brain abscesses wherever situated. But they are inconstant and variable. In some cases, they are so severe as to incapacitate the patient; in others, so slight as to be ignored. The headache is the most usual. It is generally occipital in cerebellar cases, but sometimes it is frontal.

In cerebellar abscess, disturbance of consciousness is less frequent and less profound than in temporo-sphenoidal abscess, but dullness, apathy, somnolence, loss of memory, and even coma may be observed. Curiously enough in cerebellar abscess optic neuritis is more often seen than it is in temporo-sphenoidal abscess, but it is inconstant in both.

The chief special signs indicating a lesion of the cerebellum are spontaneous nystagmus, Rombergism, deviation in the pointing test, and what is known as dysdiadokokinesis, a disturbance of co-ordinated movement on the same side as the cerebellar lesion.

The *spontaneous nystagmus* is pronounced, and it differs from the spontaneous nystagmus of a labyrinth lesion by the indefinite persistence. In the labyrinth storm, it will be remembered, the spontaneous nystagmus after the initial burst gradually subsides and disappears in a few days; in cerebellar abscess it continues as long as the lesion or the patient endures.

Rombergism.—The patient is unable to walk with closed eyes without staggering, reeling, or even falling.

On applying the *pointing test* (see p. 414) the finger of the affected side shows a deviation.

Dysdiadokokinesis may be elicited by making the patient pronate and supinate the hands rapidly. On the affected side, the movement is slow, awkward and imperfect.

Another phenomenon of the same nature is seen when the patient is made to flex hip and knee, and then suddenly to extend them.

In cerebellar lesions, the movement can only be performed slowly and gradually.

It is to be noted that these unilateral phenomena are disturbed on the same side as the cerebellar lesion.

Course and Termination.—The tendency is towards death, which takes place generally as a result of paralysis of the respiratory centre in the medulla, or from acute meningitis. The duration of the illness shows considerable variety, but roughly, it may extend for from six weeks to two months after the initial invasion. The *active* period during which the symptoms are *manifest*, and when the surgeon may intervene with most hope, usually lasts from about a fortnight to six weeks or two months. But cases may show no symptoms to attract any notice until the terminal stage.

The pressure effect of the cerebellar focus upon the respiratory centre induces, first, a dislocation of the pulse-respiration ratio during the manifest stage, and secondly the tendency to death from respiratory paralysis, which we have already mentioned.

Diagnosis.—The symptoms closely resemble those of purulent labyrinthitis; moreover, purulent labyrinthitis is frequently the precursor of cerebellar abscess, as we have seen. Consequently, it is often difficult to exclude the labyrinth. But if the hearing, as shown by the bone conduction, is not seriously impaired, then we are justified in assuming that the labyrinth is not the seat of general purulent labyrinthitis.

Cerebellar abscess and acute purulent labyrinthitis both induce spontaneous nystagmus; but in the former it persists; in the latter it diminishes and disappears in a few days, even although the labyrinthitis continues.

Sometimes the existence of cerebellar abscess is unsuspected, as when there is lateral sinus thrombosis or septic meningitis, the symptoms of which mask those of the cerebellar disease. When a lateral sinus has been operated on, the patient should be watched for signs of cerebellar abscess.

It is frequently the case that cerebellar abscess, like incipient basal meningitis, is accompanied with occipital headache and some degree of cervical rigidity. Presumably in such cases, the cerebellar focus of sepsis is inducing some localized serous meningitis (p. 553). Lumbar puncture should be performed, and the cerebro-spinal fluid examined. (See p. 557.)

Finally, it must be remembered that, in consequence of the mild nature of the preliminary symptoms, the surgeon may not have an opportunity of examining a patient with cerebellar abscess until the disease has reached the terminal stage.

Prognosis.—The prognosis of cerebellar abscess, if unoperated on, is hopeless. A certain percentage can be saved by operation.

Treatment.—As soon as cerebellar abscess is diagnosed, the cerebellum should be explored, because respiratory paralysis may set in at any moment and destroy the patient.

OPERATION FOR CEREBELLAR ABSCESS

The operation is begun by performing lumbar puncture, and removing from 20 to 30 ccs. of cerebro-spinal fluid in order to lower the intracranial pressure.

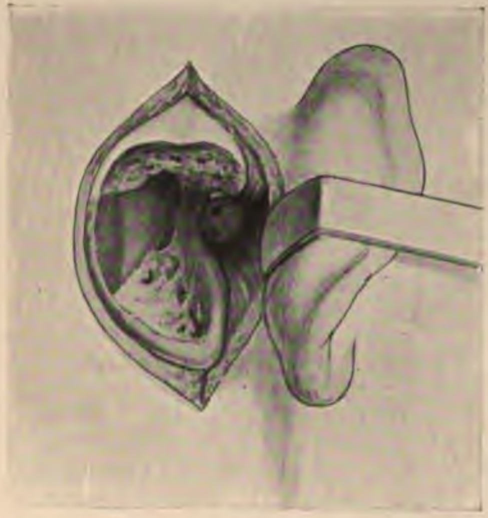

FIG. 192.—Exposure of the lateral sinus and the dura of the posterior cranial fossa, the normal procedure for effecting drainage of a cerebellar abscess, or of the meninges in septic meningitis. (Compare with Fig. 193.)

The radical mastoid operation having been performed, the bone is removed backward as in the operation for exploring the lateral sinus, and that structure is exposed. Then the bone covering the dura of the posterior fossa on the posterior face of the petrous and medial to the lateral sinus is removed up to the limits of the labyrinth. (See Fig. 192.) When a sufficient area of dura has thus been laid bare, an incision is made through it from the medial verge of the lateral sinus running inwards for the full extent of the exposed dura. This cuts across several dural vessels, and the bleeding for a few moments is free, but it stops spontaneously. After it has dried up, search is made for the abscess. A scalpel is plunged into the brain substance in a direction inward and slightly backward, to bring its point into the cerebellum just behind the saccus region. It is then withdrawn slowly, without any lateral movement, while watch is kept for pus following in its track. If this puncture proves negative, the knife is passed directly backwards; then backwards and outwards behind the lateral sinus. Sometimes the pus-searcher is used instead of, or as well as, the knife.

If the signs of cerebellar abscess have been clear, and all these punctures prove negative, the occiput behind the lateral sinus should be exposed, and an opening made through it by trephine or gouge, and after incision of the dura, the posterior parts of the cerebellum should also be interrogated.

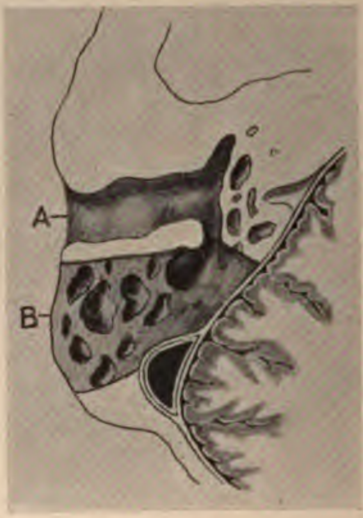

FIG. 193.—Horizontal section through the external auditory meatus (*A*), the mastoid process (*B*), the lateral sinus, the meninges of the posterior cranial fossa, and the cerebellum.

The shaded area represents the bone removed in exposing the dura of the posterior fossa. (Diagrammatic.) (Compare with Fig. 192.)

If pus is found, it emerges alongside or after the exploring instrument, and generally continues welling out commingled with brain detritus until the abscess cavity is emptied. While it is doing so, the pus-searcher is re-inserted and held in position until a drainage tube is passed into the cavity. Otherwise the cavity may be missed by the drainage tube. Two tubes are used, one alongside the other, and it is advisable to have gauze wrapped round them if there is room; or a special brain-abscess tube, made one inside the other like a tracheotomy tube, is used.

Then the mastoid wound is lightly packed with sterile gauze, and the outer dressing applied.

Unless the patient is deeply comatose, a general anæsthetic is required, chloroform being employed, and the anæsthetist, paying particular regard to the respiration in view of the risk of respiratory paralysis, keeps his patient but lightly anæsthetized.

Should respiratory paralysis supervene before the abscess is opened, artificial respiration is begun and kept up until the exploration is effected, and the abscess, if found, evacuated. Thereafter, the breathing will probably start again.

Cases are recorded of successful operation not begun until after the onset of respiratory paralysis and the cessation of spontaneous respiration.

After-Treatment.—The outer dressings are changed as often as they become soaked. The wound-packing and tubes are changed once a day. The object of a double tube is that one may be removed for cleaning while the other maintains the route to the abscess cavity. Otherwise, the cavity may be lost and the abscess re-fill.

In favourable cases, the tubes are gradually pressed out by the expanding brain, and may be removed in a week or ten days.

The results of the operation are good in many cases, but the dangerous symptoms do not always pass away with the drainage of the abscess. Some cases die comatose; others manifest an absence of reactive power and die of asthenia; others develop meningitis; while others again may succumb to a second brain abscess.

Cases which recover, however, get quite well, and are able to resume their place in the world.

During convalescence, watch should be kept for a recurrence of the pressure symptoms, and for the onset of meningitis.

CEREBRAL (TEMPORO-SPHENOIDAL) ABSCESS

Abscess of the temporal (temporo-sphenoidal) lobe differs pathologically from cerebellar abscess in lying more deeply in the brain substance, and in a tendency to reach a larger size. Clinically, it resembles cerebellar abscess in its irregular, haphazard course, but it is even more frequently latent and devoid of any localizing symptoms.

The infection reaches the cerebrum through the roof of the antro-tympanic cavity, the bone of which may show evidences of osteomyelitis, or a fistula may exist leading upwards through the adherent dura to the infected area of the brain. This track may be traceable *post mortem* through the gray matter up to the abscess cavity, which is usually situated in the white matter in the centre of the lobe. Occasionally, more than one abscess can be found.

The method by which abscess is produced in this situation is supposed to be somewhat as follows: Infection of the bone leads to adhesion of the outer surface of the dura to the diseased bone, and thereafter, the infection passing deeper, probably through infected lymphatics or venules, induces a localized lepto-meningitis which shuts off the infection from the general meningeal spaces. There follows a local infection of the underlying gray matter, which by its vascularity, according to one view, is able to shut in the infection, and to prevent abscess formation; according to another view, the vessels carry the infection through the cortical layers to deposit it in the white matter. Thus, there is formed what is known as the "stalk," leading from the antro-tympanic cavity to the abscess.

How long an abscess may remain in the brain before causing the symptoms which lead to its recognition or to death is unknown. That the period may be prolonged is suggested by the fact that many abscesses possess quite a thick dense capsule of connective tissue. But sooner or later symptoms are induced by the growing bulk of the abscess, and by the extension of encephalitis and œdema in the brain substance around the septic focus.

Left unopened, temporo-sphenoidal abscess ends in death by (*a*) bursting into the meningeal spaces on the surface of the brain, or (*b*) into the ventricles, so inducing meningitis; by (*c*) the spreading œdema and encephalitis, coupled with the ever-growing abscess, and often combined with hydrocephalus, paralysing nerve centres by a general rise in intracranial pressure; or by (*d*) inducing profound nutritional disturbances with emaciation and insuperable asthenia, phenomena which may persist even after the abscess has been opened and drained.

Symptoms.—In the classical works on otology four definite stages in the course of a brain abscess are described; the stages, namely, of invasion; of latency; of the active symptoms and of the terminal symptoms; but in actual practice it is seldom possible to effect such distinctions.

Most cases of temporo-sphenoidal abscess manifest no symptoms save headache, slowing of the pulse, and occasional vomiting, until the terminal stage of coma, when, in the absence of localizing phenomena, the otologist has to content himself with performing the radical mastoid operation, and exploring first the temporal lobe, and secondly the cerebellum. Occasionally, despite such adverse factors, the abscess is successfully opened and the patient's recovery follows.

But there is, nevertheless, a considerable proportion of the cases in which symptoms will be discovered if they are sought for before the onset of unconsciousness, and if a patient with otorrhœa complains of headache, especially if he is also having occasional " bilious attacks " with vomiting, search should be made for those symptoms.

The symptoms most frequently present belong to the phenomena of raised intracranial pressure. Thus, in addition to the headache and vomiting we have mentioned, there is in nearly all cases a regular and persistent slowing of the pulse, which varies from 40 to 60 in the minute. At the same time, the temperature, if taken regularly, will be seen to be running a subnormal course. This feature is variable, however. There may be no alteration whatever in the temperature on the one hand, and, on the other, if lateral sinus thrombosis or meningitis, or some other pyrexial complication is present, then the temperature will be raised in spite of the brain abscess.

Eye changes also—swelling of the disc, e.g., may be discovered, and when present, will add to the probability of brain abscess. But they, too, are inconstant and unreliable ; they are, as a matter of fact, more frequent in cerebellar than in temporal lobe abscess.

General disturbances in cerebral function, such as loss of memory and inability to concentrate the attention, may be observed, and a very characteristic symptom when it is present is that known as " slow cerebration," in which the whole process of thought seems to take longer than in the healthy brain ; answers to questions, for example, are correctly given, but they are slow in coming. Occasionally, more profound disturbances of mentality are induced, such as the phenomena of hysteria, or delirium with maniacal or semi-maniacal outbursts. Such symptoms as these are due to the increased pressure, and appear and disappear irregularly and without apparent reason ; but finally, unless the case is cut short by intercurrent disease or by the intervention of the surgeon, they pass into stupidity, apathy, sleepiness, and finally coma.

On the other hand, such definite brain symptoms may be entirely absent. Quite frequently, patients with temporal abscess walk into our clinics, and go about their affairs in as collected a manner as their neighbours, until, at least, the final stages are reached.

Localizing signs are less common and less definite in temporo-sphenoidal than in cerebellar abscess. But disturbances of speech, such as inability to name objects or to comprehend the meaning of certain words—phenomena which are commonest when the abscess is on the left side—point to disease in the temporal lobe. Sometimes the aphasia is pronounced.

Motor pareses and paralyses of the opposite side of the body also appear in temporal abscess, and, as Macewen pointed out, the paralysis often affects, progressively, first one side of the face, next the upper limb, and thirdly, and then only slightly, the lower limb, by reason of the fact that the nerve fibres proceeding from the cerebral cortex to those regions are progressively involved from below upwards in the spreading encephalitis and œdema around the abscess. Sensibility is not affected.

The cerebro-spinal fluid does not give us very much information. Sometimes it emerges under high pressure, but it is generally normal in appearance and constitution. Sometimes there is leukocytosis, sometimes lipoids are found, especially if the abscess is an old one, but bacteria are not present, nor is there any chemical alteration in the fluid unless meningitis has set in.

Course and Termination.—As we have already indicated, the course of temporal lobe abscess is irregular and variable; not only do cases differ greatly from one another, but the same case will show the greatest difference from time to time. One day the picture of brain abscess may be fairly distinct, next day the patient may be quite free from any symptoms whatever.

The duration of the cases is also extremely variable. I have known the symptoms of temporal lobe abscess to last for even as long as six months, and then to end in recovery after operation. But as a rule, the period during which intervention may be hopefully undertaken is to be measured by a few weeks only.

As in cerebellar abscess, the sole termination is death if the abscess is not opened and drained.

The *prognosis* in unoperated cases is therefore bad, but a considerable number may be saved by operation.

The *diagnosis* is difficult. Brain abscess shares with apoplexy; with epilepsy; with cerebral tumour; with cerebral syphilis; with meningitis, septic, epidemic, and tuberculous; with uræmia, and the infectious fevers; with alcohol and the narcotics; the tendency to induce coma. If, therefore, the patient is comatose when brought to our notice, a correct diagnosis may be impossible.

The existence of otorrhœa in a comatose patient, nevertheless, should always raise the question of an otogenic cause.

In manifest cases, brain abscess may be mistaken for brain tumour, but in the latter the progress of events is slower; eye changes are more frequent, and paralyses of the cranial nerves are common.

The presence or supervention of meningitis obscures the phenomena of brain abscess, but Politzer advises us to suspect abscess when a patient with septic meningitis is dull and apathetic instead of being irritable.

In women, the phenomena of brain abscess—altered mentality, exaggerated complaints of violent headache and periodical causeless vomiting—are liable to be mistaken for hysteria.

The diagnosis between cerebellar and cerebral abscess depends chiefly upon the presence of vestibular symptoms—Rombergism, spontaneous nystagmus, vertigo—in the former.

OPERATION FOR TEMPORO-SPHENOIDAL (TEMPORAL LOBE) ABSCESS

The radical mastoid is performed, if it has not already been done, so as to give access to the tegmen tympani and antri. The lumbar spine is tapped, and from 20 to 40 ccs. of cerebrospinal fluid is withdrawn (p. 557).

The entire bony roof of the antro-tympanic cavities is now removed with gouge and mallet so as to lay the dura bare, and search is made for granulations, a fistula, discoloration, or other signs of disease in that membrane. Note is also made as to whether the dura is tense, bulging, or pulseless.

In any case, the exploration of the brain is gone about as in the cerebellum. The dura is opened by an incision running antero-posteriorly (so as to avoid the dural vessels), and a scalpel is plunged vertically upwards into the brain and withdrawn slowly. This is repeated if negative, the direction of the knife being altered with each plunge. The depth of two inches should not be exceeded lest the lateral ventricle be opened—not necessarily a fatal accident as it happens (a distended ventricle may be opened at less than two inches).

If pus is found, the knife or pus searcher is left in position until a drainage tube can be passed alongside it into the abscess cavity. Finally, as in the cerebellar abscess, another tube is inserted alongside the first to keep the route to the abscess patent during subsequent dressings. Or, the special brain-abscess drainage tube is employed. (See p. 552.)

Then the mastoid wound is lightly packed with gauze, and the outer dressings applied.

Variations.—Some operators prefer to open the abscess through a trephine opening above the ear. A vertical incision is made from the parietal eminence to the margin of the hairy scalp above the auricle, and deepened through temporal fascia, muscle, and the pericranium to the bone. Bleeding vessels having been secured, and the edges of the wound retracted, the trephine is applied about an inch above the external auditory meatus. Or the bone may be removed, and more rapidly, by gouge and mallet, followed by cutting-bone forceps. The dura having been exposed and incised, search is made for pus in an inward direction, so as to bring the point of the knife or searcher to the brain above the tympanic region. If pus is found, the drainage is effected through the artificial opening in the skull.

Some operators, when time is not of prime importance, after exposing the dura, postpone its incision for twenty-four hours, so as to give time for adhesions to form in the lepto-meninges around the seat of operation, and to lessen the chances of meningitis. The subsequent incision of the dura and exploration of the brain, being painless, may be undertaken without an anæsthetic.

The drawback to opening the brain above the ear, and away from the mastoid wound, lies in the fact that the operation inflicts unnecessary injury on the brain. Its presumed advantage consists in the fact that the exploration is made through a clean wound, so that if it is negative there is less likelihood of infecting the brain than in operating through the mastoid wound. But as experience shows, with ordinary precautions, exploration of the brain for abscess through the mastoid wound, whether it be the cerebrum or the cerebellum that is opened, does not lead to any ill effects. Meningitis is not liable to follow, nor is brain abscess set up, if not already present.

Naturally, the mastoid wound is, as far as possible, sterilized before the dura is incised. It is a fact that the best preventive of meningeal infection from this source, is a free and not a small dural incision.

It is also recommended sometimes to make a counter opening through the squama into the abscess cavity already opened and drained from the mastoid.

SEPTIC MENINGITIS

Local meningitis, or rather meningeal effusion in the neighbourhood of a local infective focus in or about the ear, the nose or nasal sinuses, is by no means uncommon. But it is for all practical purposes negligible, as its symptoms, usually pain and headache with perhaps some sickness, disappear on the removal of the local cause.

The most important variety of the group is encountered in the meningeal symptoms (*meningismus*) of infants with acute suppuration of the middle ear before rupture of the membrane. In these patients, there may be all the early symptoms of severe meningitis—uncontrollable vomiting, head retraction, and squint, and yet as soon as the ear begins discharging, they disappear entirely. At the same time, we must not forget that these symptoms may be the prelude to genuine meningitis. Consequently, their appearance should always be taken as an indication for immediate operation, either myringotomy or the mastoid operation.

General septic meningitis, occurring in ear, nose and throat practice, presents two main varieties : the *serous*, in which the general meningeal spaces contain an excess of cerebro-spinal fluid, with leukocytes, but without bacteria ; and the *purulent*, in which the cerebro-spinal fluid does contain bacteria as well as leukocytes. For the sake of clear description, we shall take the purulent type first, and we shall also on this occasion deal with the septic meningitis which arises from the nose and nasal sinuses, as well as that arising from the ear.

GENERAL PURULENT MENINGITIS

(excluding for the moment epidemic cerebro-spinal meningitis) is caused by extension of septic infection to the lepto-meningeal spaces from a neighbouring focus. The commonest otogenic cause is purulent labyrinthitis, the septic micro-organisms finding their way into the meninges by the sheath of the auditory and vestibular nerves, and, it is said, also sometimes by that of the facial nerve. But it may also arise in consequence of lateral sinus thrombosis or of an extradural abscess (especially in saccus empyema) breaking through the dura into the meningeal spaces, and it forms the terminal event of many cases of brain abscess, when, for instance, the pus finds its way into the ventricles or out on to the surface of the brain.

Meningitis may also follow a localized osteomyelitis of the roof of the antro-tympanic cavities where the infection passes from the bone to infect the dura and subdural space. Nevertheless, it is a fact that caries of the bone in contact with the dura very seldom does lead to meningitis, as the dura over the tegmen antri is often exposed by disease without any meningeal symptoms whatever being induced.

In other words, the dura mater is the great barrier against meningeal infection, and as long as it remains intact, its simple exposure is not likely to lead to infection of the meninges.

In addition to such obvious routes of infection, cases are frequently met with in which it is impossible to determine in what manner the infection has passed to the meninges.

Meningitis arising from *nasal* sepsis is much less frequent than meningitis from an aural source, but it is known to follow ethmoidal cell suppuration by the cribriform plate becoming the seat of caries, and it also results from osteomyelitis of the cranial vault and base secondary to nasal suppuration. (See p. 372.)

In this connection, we must remark that a traumatism of the cribriform plate, or even an injury of the olfactory nerve filaments in the olfactory region of the nose is credited with a liability to meningitis by opening up nerve sheaths which are continuous with the cerebral meninges. But as with the ear, so with the nose, septic meningitis occasionally develops and it is impossible to trace the route by which the infection has reached the meninges.

As regards the bacteriology of septic meningitis, it would seem that any of the pyogenic organisms may be responsible for its occurrence.

Pathologically, the toxic infection induces an exudation into the meningeal spaces, which is more serous or more purulent according to the virulence of the infection. In the serous form, the cerebro-spinal fluid is considerably increased in quantity and turbid in character. In the purulent type, the exudate is usually of thicker consistence and may be less in quantity.

The site of origin of meningitis generally determines its clinical phenomena. Thus, infection from a nasal source, or an aural source, almost invariably leads to a meningitis of the base of the brain with symptoms, the early symptoms at all events, to correspond.

Symptoms.—The first symptom of septic meningitis is almost invariably headache, beginning in the occipital region, but spreading quickly to the frontal region, and finally involving the whole of the head. It is severe and " bursting " in character, and is made worse by movement, by stooping, by loud noises, and by light, so that photophobia is often a prominent early feature.

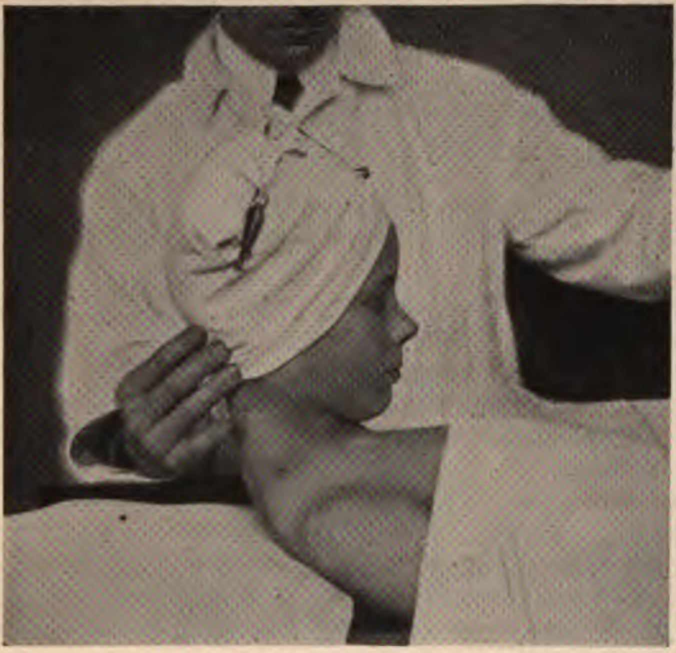

FIG. 194.—Extreme flexion of the head to elicit early cervical rigidity in incipient meningitis.

Associated with the headache is vomiting of the " cerebral " type. The temperature is raised in the early stages to about 101° or 102° F., and the pulse shows a characteristic occasional intermission or irregularity in the beat about every minute or half minute.

Even at this early stage it is possible in basal meningitis to find *cervical rigidity.* Stress is laid upon this sign, as it is a valuable early indication of incipient meningitis. The examiner's hand is placed under the occiput of the patient, who is lying on his back. The patient is then told to bend the head forward until his chin touches his chest. As he tries to do so, the surgeon's hand follows the head, supporting it, and, later, if the patient finds any difficulty in performing the act, the surgeon helps to push it forward. At the same time his fingers are palpating the muscles of the back of the neck. (Fig. 194.) Rigidity, if present, is thus at once detected, and it is a sign of great value. When there is any rigidity present, even when it is slight, the patient complains of pain in the occiput and neck on such extreme flexion of the head.

Among other early phenomena also may be mentioned *Babinski's sign*, which consists in an extension of the great toe on tickling the sole, of special value in children, and *Kernig's sign*, which is an inability to extend the knee fully when the hip is flexed.

Spontaneous nystagmus of the vestibular type, but of a minor degree, is another common early sign. Its presence may, however, be due to labyrinth irritation without meningitis.

This stage of the disease is marked also by irritability, wakefulness, and anorexia.

The stages of septic meningitis succeed each other so rapidly that no clear division can be made between them after the first period. Somnolence and finally coma follow the irritability; the pulse becomes more irregular and weak; the cervical rigidity becomes more and more pronounced until the head is fixed in the attitude of retraction and the spinal muscles of the lumbar region are contracted; the pupils, often unequal in the early stages, become irresponsive to light; paralyses of eye muscles and face appear, causing squint and often facial distortion; delirium of a noisy, violent character is common, especially in children; fixed flushes from vaso-motor paralyses are present, and the *tache cérébrale* is easily elicited.

Finally, the temperature shows a tendency to soar, and death takes place with the patient in hyperpyrexia.

The whole duration of the disease is from two to seven days, according to its virulence.

Diagnosis.—The vital importance of early diagnosis in septic meningitis, whether purulent or serous, cannot be overestimated.

For early warnings we rely upon the site and character of the headache; the presence of cervical rigidity, and of the Babinski and Kernig signs; and finally upon the examination of the cerebro-spinal fluid.

Lumbar Puncture (Spinal Puncture; Spinal Tapping).

Description.—The insertion of a long, hollow needle into the spinal theca in the region of the cauda equina and the withdrawal of cerebro-spinal fluid therefrom.

Method.—In adult patients able to walk about, the little operation may be performed in the sitting position with the head and shoulders strongly bent forward.

Otherwise, the patient is turned on one side, the head and the shoulders are bent strongly forward, the legs and pelvis being drawn upward, so as to curve the back to the utmost. With the patient in this position, a line is drawn across the lumbar spine at the level of the highest points of the crests of the ilium. This crosses the spine at the interspace between the third and fourth lumbar vertebræ. At a point one centimetre below this line and one centimetre from the middle line in adults, but in the middle line in children, a fine hollow needle, or trocar and cannula from 7 to 10 centimetres long, is thrust directly forward between the lumbar spines through the soft tissues and the tough ligamentum subflavum between the laminæ and through the dura. (Fig. 195.) The fluid is then allowed to

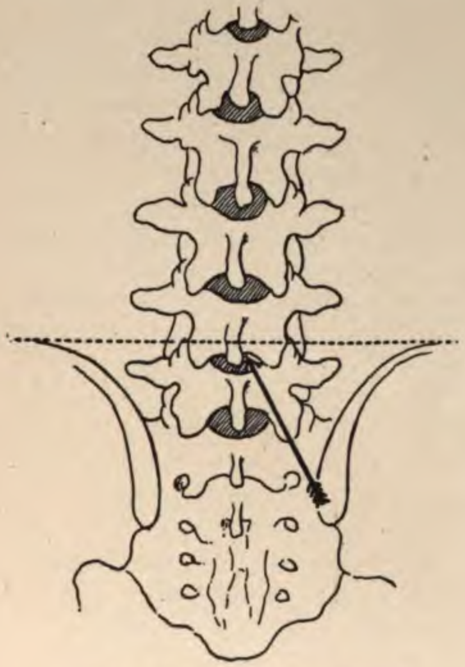

FIG. 195.—Lumbar Puncture.

escape, the first few drops, which are apt to be blood-stained, being rejected. From 15 to 30 ccs. are usually withdrawn.

The operation should be performed under strict aseptic precautions. It is not always easy to find the spinal canal, the general tendency being to pass the needle too much to one side, or to hesitate to plunge it deep enough.

Normal cerebro-spinal fluid is a colourless limpid fluid, alkaline, with a sp. gr. of 1003 to 1004. It contains no cells, and only a trace of proteid, but it does contain a definite quantity of glucose.

In all the varieties of meningitis, the cerebro-spinal fluid is, as a rule, profoundly altered. It frequently emerges from the spinal needle under high pressure; it is turbid and sometimes even flaky in appearance from the presence of cells or fibrin; and bacteria may be found in it. Chemically, also, its constitution is altered; thus its alkalinity may be reduced; it contains albumen, and it loses its glucose.

It should be noted that the first tapping is often normal.

Clinically, the condition of the cerebro-spinal fluid is often the only factor that enables us to distinguish between " purulent " and " serous " meningitis. And the distinction lies in this, that when bacteria are present in the fluid, we regard the disease as " purulent," while if bacteria are absent, we call the disease "serous." The terms " purulent " and " serous," we must add, are not to be taken literally, since the fluid tapped is always more watery than purulent, but clinically, these conventional terms are useful, as they serve to distinguish the milder from the more severe types of meningitis. That is to say, when bacteria are present in the tapped fluid, we have to deal with severe and generalized infection of the meningeal spaces, whereas when bacteria are absent, we may presume that the infection has so far not become generalized.

In other respects, as regards the cellular and chemical contents of the fluid, " purulent " and " serous " meningitis present no difference. In both, there is leukocytosis, and in both chemical examination may show the presence of proteins, and the absence of glucose.

The importance of early lumbar puncture in suspected meningitis requires, therefore, no emphasizing. For diagnostic purposes it is indispensable, and it is also of great value therapeutically, as we shall see.

In some cases we must add, and these are the most serious types of the disease, the lumbar tapping fails to evacuate any spinal fluid, by reason of the fact that it has become thick with plastic exudation. But the surgeon should remove and re-insert the needle several times before he concludes that the fluid will not run.

Examination of the cerebro-spinal fluid also enables us to distinguish between septic and epidemic cerebro-spinal meningitis, the meningococcus being present in the latter.

The presence of lymphocytes instead of leukocytes indicates some chronic cerebro-spinal process, and if meningeal symptoms are present, the likelihood is that tuberculous meningitis is the cause. In tuberculous meningitis, of course, the course of the disease is much more protracted than in septic meningitis.

From other intracranial complications of aural sepsis, meningitis is distinguished by its irregular pyrexia, and by the widespread character of its phenomena. But meningitis may be the terminal stage of brain abscess, or lateral sinus thrombosis, and its symptoms may entirely conceal the presence of the original intracranial complications.

Cerebellar abscess is generally accompanied by occipital headache and cervical rigidity, doubtless from associated meningeal effusion into the cisternæ of the base. But actual meningitis may be excluded if the temperature is normal or sub-normal.

SEROUS MENINGITIS

As we have already seen, the distinction made between purulent and serous meningitis is largely one of practical clinical convenience.

In the serous type, the infection is milder, or it is definitely localized in one area, as in the neighbourhood of the petrous in labyrinth suppuration, while the general increase in the fluid arises from the presence of toxines rather than bacteria in the cerebro-spinal spaces. And it is characteristic of the serous type that the quantity of cerebro-spinal fluid is increased, whereas as we have seen, in the more virulent types of purulent meningitis, the quantity may be lessened, and its fluidity diminished by the presence of a fibrinous exudate.

For these reasons, the *symptoms* in *serous meningitis* are less violent; the progress of events is slower; the patient does not look so ill; and the chances of recovery are greater whether the meninges are drained or not, so long as the primary focus of infection is removed. But this proviso is all-important, as the danger of a rise in the virulence of the meningitis from a general bacterial invasion of the lepto-meninges remains in existence until the source of danger is removed.

Once that is done, many cases of serous meningitis tend to spontaneous recovery, although most modern surgeons prefer to remove all possible risks by free drainage of the meninges.

Prognosis of Septic Meningitis.—As we have already remarked, the prognosis of serous meningitis is not unfavourable if the primary focus is removed, and if the symptoms undergo moderation in their severity after lumbar puncture.

In the purulent type, the outlook is much more grave, but modern operators have succeeded nevertheless in materially reducing the mortality. A rapid progress; inability to obtain a free flow of cerebro-spinal fluid from the spine and from the focal region in the cranium; and the appearance of profound septic poisoning in the aspect of the patient are ominous signs.

It is no exaggeration to say that the prognosis is good or bad, according as the flow of cerebro-spinal fluid is free or scanty.

Treatment of Septic Meningitis.—In both types, the treatment is the same. First, remove the infective focus;—the radical mastoid operation is performed, and if necessary the labyrinth is opened and drained; extradural abscesses are evacuated; and if the symptoms raise the suspicion of lateral sinus thrombosis or of brain abscess, exploration of these regions is also undertaken.

Secondly, the *meninges are drained.*

DRAINAGE OF THE MENINGEAL SPACES

There are several methods by which this may be accomplished. First, the lumbar spine is tapped once or twice a day or even oftener, as much cerebro-spinal fluid being run off as can be obtained. Indeed, it is often advisable to leave the trocar *in situ* so that continuous spinal drainage may be secured. Experience has shown that there is little or no risk of excessive depletion of the cerebro-spinal system. The spinal tapping has the effect of draining the whole of the cerebro-spinal fluid-filled spaces, and no further drainage may be necessary.

Nevertheless, the author advises and practises drainage of the meninges in the neighbourhood of the primary focus of infection, as well as spinal tapping, since by doing so the chief seat of meningeal infection and reaction is opened. In the case of the ear, there are two methods of effecting this.

Drainage through the Meninges of the Posterior Cranial Fossa—After the radical mastoid has been performed, the lateral sinus is exposed, as detailed on p. 538. Then with gouge or cutting bone forceps, the dura of the posterior fossa is laid bare, as in the operation for cerebellar abscess. (Fig. 192.) It is advisable, however, in the present circumstances to carry the removal of bone further in towards the internal auditory meatus, as this brings us near to the saccus endolymphaticus, and also to the cisterna magna at the base of the brain.

This done, the dura is incised from within out along the whole extent of its exposed area, and the flow of cerebro-spinal fluid, which is often free and continuous, is encouraged by a drain of gauze or even a small rubber drainage tube passed through the inner end of the dural incision towards the cisterna magna.

The second method of meningeal *drainage* is effected *through the labyrinth* and the internal auditory meatus, and it is the route of choice when the labyrinth is opened for labyrinth suppuration (West and Scott).

After the labyrinth operation has been performed (Fig. 188), a fine curette or burr is passed through the anterior opening into the labyrinth to the modiolus, and this thin plate of bone is broken down so as to give access to the internal auditory meatus. Success is betokened by the welling up of cerebro-spinal fluid into the wound.

This last method should not be adopted if the labyrinth is active, and if the drainage through the dural incision is plentiful, as it necessarily destroys the hearing in that ear.

After-Treatment.—If the discharge of cerebro-spinal fluid is free, the outer dressings soon become soaked, and will require frequent changing. A light gauze packing occupies the mastoid wound, and will require changing once a day. The patient should be encouraged to lie on the homo-lateral side to aid the drainage.

In all brain cases, where the movement of the head causes suffering, a many-tailed bandage cut to fit the head is a great saving of pain and distress in the changing of dressings.

Results.—Meningeal drainage has reduced the mortality of all kinds of septic meningitis by about thirty per cent., the mortality now being about fifty per cent

Improvement is first manifested in the patient's aspect and demeanour. He looks better and brighter. Then the signs of brain irritation, such as somnolence and delirium, become less obvious, and the headache and its associated expression of anxiety disappear. The pyrexia and cervical rigidity are the last symptoms to pass off, the temperature remaining above normal for several days after the other symptoms have disappeared. The flow of cerebro-spinal fluid continues for several days after the operation. If, after it has ceased, a recurrence of fever and headache set in, a timely spinal tapping is often sufficient to prevent any further adverse developments.

Recovery, when it takes place, is complete so far as the brain is concerned. There is no mental weakness left.

The *Treatment of Rhinogenic Meningitis* is the same in principle as that of the otogenic disease, the only difference being that in the case of the nose the primary focus to be removed lies usually in one of the sinuses, ethmoidal, or sphenoidal, for which see p. 314. Or, if the primary disease is diffuse osteomyelitis, the bone must be removed as described at p. 374.

Drainage of the cisternæ may be then instituted through a mastoid operation undertaken for the purpose, without, of course, opening up the ear cavities.

CHAPTER XIII

THE CHRONIC CATARRHS OF THE MIDDLE EAR AND OTOSCLEROSIS

Under the appellation of "Chronic Catarrh of the Middle Ear" is grouped a large variety of pathological conditions, which probably represent a number of different lesions, some of them, no doubt, infective or bacterial in origin; others "constitutional," that is to say, induced by some toxic influence generated elsewhere in the body than in the middle ear cleft.

Pathologically, the changes include subacute or chronic sero-mucous inflammation of the mucosa of the Eustachian tube and middle ear; adhesive catarrh of the middle ear, and atrophic catarrh of the middle ear.

Clinically, the symptoms are very much the same for all, varying only in their rate of progress. There is deafness of the obstructive type, tending to get worse with time; there is tinnitus, more or less constant and severe; and there is an absence of any discharge through the membrane.

Thus, the distinction between the varieties of this group is not always easy to effect, and the difficulty is further increased by the fact that very similar symptoms and signs characterize the disease known as *Otosclerosis,* in which the mucous membrane is free from pathological changes, but the foot-plate of the stapes becomes fixed in its oval window by bony ankylosis.

Consequently, in the divisions of the subject adopted here it must be understood that we do not claim to do any more than to describe certain group-tendencies as manifested in the clinical and pathological phenomena at our disposal.

We proceed to consider these groups in detail.

SERO-MUCOUS CATARRH OF THE MIDDLE EAR (SUBACUTE CATARRH: MOIST OR EXUDATIVE CATARRH)

In this type of catarrh, the middle ear presents a certain degree of active inflammation, some hyperæmia of the mucosa is present, and there is a tendency to the exudation, and, in some cases, to the retention and accumulation of a sero-mucous secretion in the tympanum.

Etiology.—The disease is a frequent accompaniment of acute and chronic hypertrophic catarrhs of the nose and throat, and is, therefore, favoured by the presence of adenoids; or of chronic rhinitis from whatever cause arising. (See p. 265. See also Eustachian catarrh, p. 463.) In such conditions we find the ear disease tending to recur and to pass insensibly into a chronic catarrh of the middle ear, which may end in atrophy or in the development of adhesions within the tympanum.

Again, a subacute exudative otitis media attends the active stages of measles, scarlet fever, and diphtheria, but this type is non-recurrent, and is, on the other hand, more likely to discharge through the membrane and so to come under the category of acute purulent otitis media.

Thus, sero-mucous catarrh of the ear is probably always due to local bacterial infection.

Pathology.—The site of the catarrhal inflammation varies. In most cases it seems to affect the whole middle ear cleft, but in some the chief seat is located in the Eustachian tube, while in others the tympanum alone seems to be affected. The quantity of the exudate varies considerably from time to time in accordance with the fluctuations in the catarrh and the permeability or stenosis of the Eustachian tube, and as the disease gets well or passes into the more chronic types, so does the exudate tend to diminish in amount.

Symptoms.—The disease is characterized more than the other forms of catarrh by unpleasant sensations in the ear. A feeling of fullness with numbness in the side of the head, "as if the ears were stuffed with cotton-wool" is a common complaint, and it is remarkable that the patient has the impression of being much more deaf than he actually is. Another annoying subjective symptom is *autophony*, which consists in the patient hearing his own voice unpleasantly loud. It is most noticeable when the disease is unilateral, and it is occasionally accompanied by a disturbance in the hearing for musical tones—notes sounding flat in the affected as compared with the normal ear. Hyperacusis, often painful, for shrill or loud sounds is common, due, doubtless, to myopathic interference with the stapedius muscle, which, moreover, sometimes manifests hyperexcitability, the normal "stapedius hum" audible on stroking the cheek being replaced by a sudden loud roar or crash.

Tinnitus is usual. It is low-pitched and variable in intensity, and it tends to disappear after treatment, at all events in the earlier attacks.

Crackling in the ear on blowing the nose, or during swallowing, yawning, and other movements of the throat is common. The *deafness* is of the obstructive type, and varies from hour to hour, and sometimes even when the position of the head is changed. Unlike the deafness of chronic dry catarrh, it is worse in the morning.

Examination of the membrane with the speculum reveals, sometimes a slightly congested membrane; sometimes a retracted membrane (see p. 424); sometimes, again, fluid is visible through it as a dull shadow, the upper level of which can be seen to move as the patient moves his head. (See p. 464.)

By means of the diagnostic tube, we may be able to hear, during inflation, the moist râles produced by the bubbling of air through the exudate.

Course and Termination.—The condition is transitory, its presence or absence depending upon the course of the associated catarrh, acute, sub-acute, or recurrent, in the nose and nasopharynx. Its first attacks tend to disappear entirely with complete restoration of function, but, if the nasal or nasopharyngeal disease persists or recurs, so does the aural malady become more and more fixed until finally the disease passes into definite atrophic or adhesive catarrh of the middle ear with gradual progressive lowering of the hearing power.

A common history in these cases is that the deafness begins with a succession of such attacks as we have just described, accompanying acute or sub-acute nasal catarrh, to which the patient has been very liable in early life. As time passes, the nasal catarrh—"the tendency to bad colds"—entirely disappears, but the deafness persists and gets progressively worse.

The *Diagnosis* presents no difficulties. But a distinction should be drawn between the middle ear exudate due to catarrh, and a rarer form due to passive congestion from stenosis of the Eustachian tube.

Prognosis.—If the causative nasal or naso-pharyngeal disease can be cured, the prognosis of exudative catarrh of the middle ear is good as regards non-recurrence and function. But this only applies to the early attacks; later, there is a tendency, which becomes greater and greater as time goes on, for the ear disease and the deafness to persist and to progress independent of the nasal disease. In any case, however, the unpleasant subjective phenomena disappear, and do not return.

Treatment.—(*a*) During the attack the treatment is that of acute nasal catarrh. (See p. 261.) The patient, especially if the weather is cold or damp, should be kept indoors in an equable temperature.

Locally, the ears should be well inflated every two or three days with Politzer's bag, as this opens the Eustachian tube, and aids in the expulsion of the retained tympanic exudate.

After the subacute symptoms have subsided, the catheter may be used as already detailed on p. 432. But it should be avoided during the acuter stages.

The orifices of the Eustachian tubes, which the nasal endoscope often shows to be red and swollen (Plate II., Fig. 1), should be painted with argent. nitrat. grs. xx. to ℥i aq.; or with zinc chloride grs. xx to ℥i. Aq.

If the Eustachian obstruction persists in spite of treatment directed to the nose and naso-pharynx, the same solution may be applied to the interior of the tube by means of a suitable applicator.

Sometimes the liquid exudate in the tympanum is slow to disappear in spite of measures directed to clearing the Eustachian tube. When that is the case, recovery may be hastened by an incision through the membrana tympani, as in acute suppuration (see p. 476), the viscid secretion being aspirated out of the tympanum by means of a Siegle speculum. Strict asepsis is necessary for this procedure, and after-treatment with antiseptic drops is advisable. (See p. 491.) Otherwise, it is safer to refrain from opening up the tympanum.

(*b*) After the attack has subsided, active steps should be taken to reduce the tendency to nasal catarrh. This is the stage and these are the cases in which the nasal and naso-pharyngeal treatment of deafness is most likely to be successful. Adenoids and tonsils should be removed; deflected septa rectified; turbinal hypertrophies reduced; suppurating nasal sinuses laid open; and, if necessary, anti-catarrhal vaccines given.

Among other abnormalities which should be removed are the small clumps of adenoid tissue frequently present in the fossæ of Rosenmüller. (See p. 382.) These form a favourite breeding-place for attacks of catarrh, and their proximity to the orifice of the Eustachian tube renders them a source of trouble in sero-mucous catarrh of the middle ear. They are often found even when the naso-pharynx otherwise is clear of adenoids.

Once the stage of sero-mucous catarrh is past, and that of adhesive or atrophic catarrh entered, nasal operation and treatment are disappointing. A single attack of sero-mucous catarrh of the middle ear, or as it is popularly named, "deafness from cold," should, therefore, be regarded as a danger-signal, and every effort should be adopted to prevent its recurrence.

In addition to these local measures, the patient's habits and life will require regulation. Occupations exposing him to extremes of weather should, if possible, be given up; he should carefully avoid getting wet; sleeping with open windows is unsuitable, however commendable that habit may be in general; and local irritants, such as tobacco and alcohol, should be avoided.

This treatment and regimen may seem to be severe, but the alternative is chronic catarrh of the middle ear and progressive irremediable deafness, and sufficient emphasis is not always laid upon this fact by otological authorities.

CHRONIC ADHESIVE CATARRH OF THE MIDDLE EAR

(Chronic Atrophic Catarrh)

In this condition we are often dealing with the end-results of neglected sero-mucous catarrh, in consequence of which the tympanic lining, having passed through a stage of infiltration, now undergoes cicatricial change with the usual contractions, adhesions, and distortions consequent upon the formation of scar-tissue.

As time goes on, the ossicles are interfered with, and their movements are impeded by the formation of cicatricial bands; by their displacement and fixation; and often by adventitious adhesions, such as that between a markedly indrawn malleus handle and the promontory. Such changes naturally produce deafness by hindering the ossicular transmission of sound waves.

In these changes, also, the mucosa in and around the labyrinth windows participates, and this factor, by limiting their flexibility, tends still more to the induction of deafness.

We have thus far laid stress upon chronic catarrh of the middle ear as a secondary effect of sero-mucous catarrh, but it is necessary also to state that these adhesive and contractile changes as frequently, or more frequently appear without any antecedent catarrh whatever, or at all events without any history of such catarrh, either in the nose or in the ear. The onset is insidious.

In other words, in very many cases the cause of the disease is unknown. Generally speaking, it is ascribed to such depressing conditions as rheumatism, gout, cold, alcoholism, or Bright's disease; and no doubt such diseases may predispose to chronic catarrh of the middle ear, but the reason why one rheumatic person develops dry deafness and another does not, is unknown. Even heredity does not help us, as in most of these cases there is no family history of any deafness whatever.

Certain more recent speculations may be given for what they are worth. According to one view, these middle ear changes may be the after-effects of slight acute or sub-acute catarrhs in infancy and early childhood. According to another view, the changes may be due to some constitutional toxic process; while Watson-Williams has recently suggested that the ear changes are secondary to bacterial infection, not necessarily causing suppuration, in the sphenoidal and other nasal accessory sinuses.

Symptoms.—The prominent symptoms are tinnitus and progressive deafness.

The tinnitus is continuous and persistent, varying, however, in loudness and sometimes also in quality from time to time. It is usually worse in cold, damp weather, and when the patient is tired, and is most objectionable when the environment is peaceful and still, as it then is most obtrusive. Such patients never realize the sensation of quietness, and the continual harassment of never-ceasing tinnitus may, indeed, end in neurasthenia or even insanity (p. 415).

In quality, the tinnitus varies. It is described as a hissing "like steam escaping,"; or a noise like the sea on the beach; or it may be low-pitched and roaring. It seldom prevents sleep, but occasionally wakes up the patient at night by sudden intensifications.

The deafness in chronic catarrh of the middle ear, is, in the beginning, at all events, obstructive in type, and it tends to get worse as time goes on, in defiance of treatment. It is often the case that the tinnitus precedes the appearance of deafness, and that as the deafness gets worse, the tinnitus gradually becomes more and more subdued. There is, however, very considerable variation in the rate of progress of the deafness. Some cases become seriously deaf to the voice within two or three years from the onset of the tinnitus. In others, again, the malady seems to be stationary for prolonged periods. The younger the patient is when the disease sets in, the more likely it is to end quickly in grave loss of hearing.

From the hearing tests one or two hints may be obtained with regard to prognosis. Thus, a loss of hearing for the watch out of proportion to the loss for the medium tuning forks is generally regarded as ominous. And so also is a falling off in the bone-conduction.

When this last takes place, we may find that the tests begin to show mixed obstructive and nerve deafness, and this is a change which is prone to appear sooner or later in all old-standing cases. When it does occur, it is almost inevitably accompanied by an increase in the rate of downward progress. We may suppose that in consequence of the middle ear disease, and perhaps also as a result of its own diminished activity, the cochlea undergoes degenerative changes.

By the time this stage is reached, the deafness has, as a rule, become very severe. The hearing of even loud conversational tones is lost, and artificial aids are needed to keep the patient in touch with the world of sound. But a modicum of hearing is nevertheless, always retained. These patients seldom become absolutely deaf.

The other common ear symptoms such as pain and vertigo are not, as a rule, prominent. Occasionally, twinges of pain are felt, and attacks of vertigo, generally mild, sometimes severe, are experienced.

Finally, it ought to be understood that although subjective sensations of fullness, autophony, and so on, are commoner in the exudative type of catarrh, nevertheless, they are occasionally also found in chronic atrophic catarrh.

On examination, the condition of the tympanic membrane in chronic catarrh of the middle ear is sufficient to enable us to base our diagnosis upon it alone, but it must be remarked that the state of the membrane gives no certain indication as to the degree of deafness reached.

The membrane is nearly always in the position of retraction and its texture shows great alteration. Either it is dull, lustreless, opaque and thickened, or it is thin, fine, membranous and even transparent. Or, again, it may be thick in some points and atrophied in others, with dense white islets of calcareous deposit. Sometimes it is so attenuated that the ossicles and inner tympanic wall are visible through it, and it is closely wrapped around the short process and neck of the malleus. Siegle's speculum often shows the membrane and the tip of the malleus handle firmly adherent to the promontory, in which case we may take it that the ossicular chain is immobilized. In others, the thin membrane flaps loosely in and out as the air-ball is compressed.

This naturally means deafness, but so long as the stapes remains movable in the oval window, the deafness is only moderate. Should the stapedio-vestibular articulation, however, become stiffened or *à fortiori* ankylosed, the deafness will be very great. Interference with the mobility of the stapes in chronic catarrh results from the envelopment of the foot-plate of that ossicle in scar-tissue, and this may, in time, become ossified, in which case we have one of the varieties of what is known as " otosclerosis."

In a proportion of cases the cicatricial changes affect also the Eustachian tube, producing stenosis of a more permanent and obstinate character than that found in exudative catarrh.

Diagnosis.—The diagnosis of chronic middle ear catarrh seldom presents any difficulty, but it may be easily confused with the classical otosclerosis.

Prognosis.—The variable progress of the disease renders the prognosis often difficult. But, speaking generally, the prognosis in cases of middle-ear catarrh depends, firstly, upon the presence or absence of signs of labyrinth implication, and, secondly, upon the results obtained on inflating the ears.

In the sero-mucous variety, when there is Eustachian obstruction, inflation, both by Politzerization and by the catheter, will be carried out with difficulty; but when accomplished, the opening up of the tube and tympanum will result in an improvement in the hearing. On the other hand, in atrophic catarrh, inflation is easy, and as a rule, no improvement follows. Improvement as a result of inflation is thus a favourable omen.

Treatment.—Once chronic middle ear catarrh has definitely set in, all we can expect of treatment is to arrest or to delay the progress of the malady There is no known method of curing established deafness that is due to fibroid changes in the middle ear.

In the earlier stages, regular courses of treatment by catheterization are decidedly beneficial. The catheter should be passed once or twice a week, and menthol or iodine in liquid paraffin injected. (See p. 465.) This should be continued for a month or six weeks, and then interrupted again for three or four months.

Politzer's method of inflation may be tried, but it is generally useless, and indeed may be harmful. If after this kind of inflation the hearing is found to be worse, it should not be tried again. We are no longer dealing with a lowering of the air-pressure in the middle ear, but with hard, fibrous bands and dense adhesions.

The nose and throat should be made healthy, but unless there is a definite local reason for nasal or other operation, such should not be performed in the hope of curing, or of even arresting the middle ear disease.

Efforts directed at securing good bodily health are really of more importance. Exposure to wet and cold; residence in damp localities; over-indulgence in rich food, in alcohol, in tobacco; and, on the other hand, poverty and insanitary surroundings, all exercise an evil influence in middle ear catarrh, and should be avoided, as far as possible.

Those who are able should winter in the inland districts of Algiers or Egypt. The sea-coast and low-lying localities generally are bad, but dry, mountainous or desert air is beneficial.

Internally, tonics may be tried, but I have seen when it is well tolerated more benefit from small doses of iodid. of potass long continued than from any other drug.

A course of blisters on the mastoid is still advised by some otologists.

OTOSCLEROSIS (ANKYLOSIS OF THE STAPEDIO-VESTIBULAR ARTICULATION—ANKYLOSIS OF THE STAPES.)

This name (literally *ear-hardening*) is generally applied to cases of bony ankylosis of the foot-plate of the stapes in the oval window. As we saw in the last section such ankylosis may occur in the old fibrous tissue of chronic catarrh, but this is not considered to be the " true " otosclerosis.

In "true" otosclerosis there is said to be no catarrh or other inflammatory change in the mucosa of the middle ear, the only visible change being an osteoporosis of the bone of the labyrinth, in consequence of which its dense, ivory, or "petrous" bone is replaced in certain areas by bulky, spongy bone. This change is most pronounced in and around the cartilage and bone of the oval window, where osseous outgrowths and trabeculæ forming between the margin of the oval window and the stapes, lead to the fixation of that ossicle. Pathologically, these appearances resemble very closely those of rheumatoid arthritis (J. O'Malley), but otosclerosis does not seem to be specially common in patients with rheumatoid arthritis of the other articulations, nor do patients with otosclerosis show, as a rule, signs of rheumatoid arthritis elsewhere than in the stapedio-vestibular articulation.

The changes we have described do not seem to affect the mucous membrane of the middle ear. It is to all appearance healthy. Consequently, many authorities regard this form of stapedial ankylosis to be a disease of the bone of the petrous; unrelated to any disease in the middle ear. Recently, however, J. S. Fraser has shown that bony ankylosis of the stapedio-vestibular articulation may follow purulent otitis media, a finding which certainly suggests that even "true" otosclerosis may be initiated by inflammation of the tympanic mucous lining. But while that may be true, it is also unfortunately true that we cannot tell why ankylosis occurs in some inflammatory cases and not in others.

The cause of the disease is, therefore, not yet known. It seems to affect women oftener than men, the most distressing cases being young women in their twenties, but it may begin at any age and attack either sex. There seems also to be a real hereditary tendency to the disease.

Debilitating illness, especially such as entail severe loss of blood, and the various forms of anæmia are all known to precipitate otosclerosis, and it is particularly true that childbirth is very apt to lead to the disease or to aggravate otosclerotic deafness if that already exists.

Practically speaking, the importance of otosclerosis lies in the fact that it is incurable, and that the deafness it causes is progressive to the point of very grave impairment, though not to absolute loss of hearing.

Symptoms.—Tinnitus is the first symptom to appear. It is continuous, and, in typical early cases, high-pitched and simple, but often in the later stages it becomes compound. (See p. 415.) After a period of several months deafness sets in and becomes progressively worse, sometimes leading to a grave loss of hearing in from two to three years; sometimes proceeding much more slowly, and even at times manifesting halts in its progress. The deafness at this period is of the true obstructive variety. But after the lapse of some years signs of nerve deafness usually make their appearance; the hearing for high tones is reduced; and thereafter the deafness becomes rapidly worse, although in this, as in severe chronic catarrh, the deafness does not become absolute.

The following are the signs upon which we rely in distinguishing otosclerosis from those varieties of obstinate and serious deafness due to fixation of the stapes from the presence of cicatrices, bands, etc., in chronic catarrh or residual suppuration. On examination with the speculum in otosclerosis it will be seen that the obstructive deafness is not caused by any disease of the tympanic lining, for the tympanic membrane appears to be quite normal. There is no alteration in texture, no distortion of the light-reflex, and no change in colour, save that a faint rosy glow of "flamingo-red" may be seen in the postero-central region of the membrane, reflected, it is said, from a red and congested promontory. Further, we find that the deafness is not due to Eustachian obstruction, for on Politzerization the tubes are discovered to be perfectly patent, while no improvement follows the inflation. Finally, we consider the typical picture to be complete when paracusis Willisii is present.

Summing up these points, the symptoms of otosclerosis are: a high-pitched tinnitus, severe and often irremediable deafness —in the first stages obstructive in character, in the later stages labyrinthine. The tympanic membrane is healthy and the Eustachian tubes patent. It is distinguished from chronic catarrh and residual suppuration by the absence of the appearances of tympanic disease, present or past.

It is obvious that this distinction cannot always be clearly obtained. There is no reason why persons who are already the subjects of disease in the middle ear should not also suffer from otosclerosis; and thus it comes that we are very often confronted with cases in which grave obstructive and nerve deafness is combined with evidence of disease in the tympanic lining, and in which it is impossible to determine whether the deafness is due solely to the middle ear disease or to its association with otosclerosis.

In this connection we should mention that there is a common class of case which generally goes by the name of chronic catarrh of the middle ear, but which, on account of its characters, should be included in the otosclerotic group.

Its features are distinctive. The tympanic membrane is uniformly atrophied, and shows indrawing, but its movements, when tested by the Siegle speculum, are, at all events in the early stages, quite normal.

Tinnitus is a prominent symptom. But the outstanding feature in the case is, as in the classical otosclerosis, the fact that the patient does not suffer, and never has suffered, from any catarrhal or other nasal or naso-pharyngeal disease. The nasal septum is straight; there is no history of obstruction; the turbinals are well-formed; the naso-pharynx is free from any indication of adenoids past or present; the Eustachian orifice is patent—often unduly patent; the contour of its lips is firm and prominent; and there is no sign anywhere of any catarrh, inflammation, or œdema.

Indeed, the mucous and submucous tissues not only of the nose and naso-pharynx, but also of the pharynx and soft palate show some atrophy and a definite but not a pronounced paleness, such as we are accustomed to see in old people who are in all respects healthy. This is obviously a senile change, and when it is seen, as it not infrequently is, in the young or middle-aged, and is in them associated with tinnitus, obstructive deafness, and atrophied tympanic membranes, experience teaches us to regard the combination as ominous in so far as the hearing is concerned.

The absence of any rosy reflex on the membrane, and the fact that that structure shows atrophy and indrawing, may be held as distinguishing this class from that of the classical otosclerosis. Another point of difference is, that in spite of a gaping Eustachian orifice the tube is occasionally so narrow as to prevent inflation of the middle ear by the catheter.

Nevertheless, the slow and irresistible advance of the obstructive deafness in this type of deafness leaves no doubt that the essential lesion is a fixation of the foot-plate of the stapes as in the genuine classical otosclerosis.

Treatment.—The only treatment worthy of trial in otosclerosis is the internal administration of potass. iodid. in moderate doses over prolonged periods.

Albert Gray advises the treatment of constipation, if it is present, by paraffin, in order to remove any intestinal toxic influence. He also reports occasional benefit to the ear symptoms from iron internally in apparently true otosclerosis associated with anæmia.

Local treatment by the catheter and by inflation seems to be quite unavailing in genuine otosclerosis, and attempts to improve the hearing by nasal or other operations are worse than useless, since the operation is very liable to be followed, as child-birth is, by an increase in the deafness.

Practically speaking, the difficulty in diagnosing genuine otosclerosis—or in other and more accurate words, the difficulty in diagnosing the existence of bony ankylosis of the foot-plate of the stapes, has led to the following rough rule of therapeutics: if after using the catheter the hearing is improved, local treatment by the catheter should be continued, and efforts made to reduce or cure nasal obstruction and catarrh. But if there is no improvement after the use of the catheter, local and nasal or naso-pharyngeal treatment is not likely to be of any value whatever. (For the treatment of the severer grades of deafness, see p. 589.)

CHAPTER XIV

NERVE DEAFNESS AND LABYRINTH DISEASE

We have already dealt with the subject of suppurative labyrinthitis (see p. 522), and have alluded, in passing, to the tendency in non-suppurative middle ear diseases for the labyrinth to become involved. Ménière's syndrome or "the labyrinth storm" has also received attention (p. 417), as has the interesting herpes oticus, and there are only left for us now to discuss certain aspects of cochlear and vestibular disease hitherto not dealt with.

The diagnosis of the nature and of the situation of the lesion in cases of nerve or perceptive deafness is often a matter of difficulty. It is true that when perceptive deafness is associated with old-standing middle ear diseases like chronic catarrh and otosclerosis, we may assume with some confidence that it is in the cochlea that the lesion causing the nerve deafness is situated, and that the cochlear disease is induced by that of the middle ear. This view is supported by what is known of the pathology of these diseases. In old-standing chronic catarrh, the organ of Corti frequently shows signs of degeneration in lymphocytic infiltration, and in disappearance in parts of the specialized epithelium, while in otosclerosis the bone changes may extend to the wall of the cochlear canals, and so lead to slow destruction of the cochlear organ.

Again, when in conjunction with the signs of nerve-deafness we find symptoms and signs of vestibular involvement; if, for example, the nerve deafness is associated with a history of one or more definite attacks of vertigo, both violent and prolonged; or, if the nerve deafness is associated with an absence of or an impairment in the vestibular reactions, then also we may assume that we are dealing with a lesion located in the labyrinth, or somewhere along the course of the auditory and vestibular nerves before they separate from each other on entering the pons.

Moreover, when perceptive deafness is associated with the signs of syphilis, and especially of syphilitic disease of the meninges of the base of the brain with, it may be, paralysis of others of the cranial nerves, the cause of the nerve deafness may be, for all practical purposes, admitted. So also in tabes, when its onset corresponds in time with the other symptoms of that disease.

When combined with, or followed by vestibular impairment and disturbance and the signs of intracranial tumour, with, it may be, facial paresis or paralysis, the diagnosis of cerebello-pontine tumour involving the acoustic nerve will occur to us.

Thus the existence of nerve deafness in a patient raises a large number of neurological questions, which necessitate in all cases a complete " neurological survey " of the case, but it not infrequently happens that in spite of all diagnostic efforts the real cause of the nerve deafness remains hidden from us.

The relationship of local vestibular disease to local cochlear disease has not yet been completely settled, and the results of researches to hand so far are not in agreement.

The writer's results will be given under the various diseases dealt with.

That the two systems should frequently be associated in disease is no more than we should expect from the anatomical relationship of the two organs ; from the fact that each is permeated by a system of canals which is in direct continuity with the other ; and from the fact that in the Ménière syndrome, when a sudden pathological event disorganizes one organ, the other also is involved in the disturbance.

We proceed now to discuss in detail the known labyrinth diseases, taking the more common first.

NOISE DEAFNESS

Under this appellation we include the nerve-deafness which results from prolonged exposure to noise in the course of occupation. Any painful noise if long-continued may be followed by deafness, and in addition to that, temporary and even permanent nerve deafness may follow a single exposure to overwhelmingly loud noise, such as that of an explosion.

This type of deafness, therefore, seems to be due to the injurious action of an overpowering stimulus, either single or repeated, upon the organ of Corti, as signs of degeneration are visible in it. An explosive noise, coupled as it is with a massive physical displacement or push of the atmosphere, may, however, rupture the tympanic membrane (see p. 459), and the occurrence of this injury seems, at times, to have the strange effect of protecting the deeper cochlea from damage, since when the membrane has been ruptured, nerve deafness is often less severe and prolonged than it is after exposure to an explosion in which the membrane has escaped.

Continual exposure to the deleterious noises of industrial life has no obvious influence upon the conducting apparatus, but leads, as we have said, to permanent nerve deafness.

Among the occupations liable to induce noise deafness are artillery firing, naval gunnery—the smaller guns with rapid fire being particularly blamed; boiler-making; engine-driving; printing; engineering and metal work generally; and so on.

Individuals vary considerably in susceptibility to the evil effects of noise; and middle ear disease, if it impairs the muscular accommodation of the conducting apparatus, so lessening the protection of the cochlea against the effect of noise, will probably predispose to noise-deafness.

On the other hand, we may assume that the interference with the conducting apparatus produced by some forms of middle ear disease may actually protect the cochlea against loud noises. But all these considerations are invalidated by the fact that the cochlear destruction produced by noise may, in many cases, be effected by the transmission of the deleterious sounds through the bones of the body and skull, as much as, or more than, by their conduction through the air and the middle ear.

Symptoms.—The onset is often noticed after the first exposure to the noise, but the first effects would pass away were it not that the exposure is repeated, and in this way the deafness tends to become permanent. Tinnitus is present immediately after exposure, and in the early stages, but it disappears early, and is not, in uncomplicated cases, a prominent symptom.

After reaching a moderate degree, the deafness for years remains stationary, or advances but slowly. This is particularly noticeable when the patient abandons his noisy environment, but the deafness is, nevertheless, permanent.

In my findings, the vestibular reactions have been impaired in noise-deafness, the explanation probably being that the vestibular end-organ may be stimulated by certain sounds as well as by changes in position. If, that is to say, the vestibular organ is stimulated by sound, it may also, like the cochlea, be damaged by excessive sound.

In this supposition there is nothing inherently improbable. It is supported, moreover, by the following considerations:

Vertigo is occasioned at times by loud noises.

The cochlea and the semicircular canals are occupied by a common system of closed—or, at all events, of tense fluid-filled canals.

The foot-plate of the stapes lies as near to the sensory epithelium of the canals as it does to that of the cochlea.

Further, sensitiveness to sound on the part of the vestibular organ would obviously have a protective function, since by the direct control the vestibule exercises over the skeletal muscles, their stimulation and contraction would ensue upon the reception by the vestibular end-organ of a sudden sound more promptly than if such stimulation had to travel by way of the hearing centres in the temporal lobe to reach the motor neurones in the cord.

Personal observation, also, shows that the act of "starting" at a sudden loud sound takes place in time a little antecedent to the reception and acknowledgment of the sound by the consciousness—a point which clearly supports the above reasoning (P. M'Bride).

Treatment.—People exposed to noise in the course of their avocations should plug the meatus with some material relatively impermeable by sound, such as cobbler's wax, or plasticine; or the ear-plugs made by the instrument-makers should be worn at their occupation, the plug being inserted before exposure to the noise. Cotton-wool plugs, though popular, are practically useless.

This device only confers a partial protection, however, as the sound is able to reach the cochlea through the bones of the body and head, but even so, this slight protection is better than none.

Naval and military gunners, in addition to meatal plugs, learn to cover their ears with their hands, and to stand where the sound is least heard. On board ship, the gun crew should stand on rubber or cork mats with knees and hips bent, so as to break the rigid continuity of the skeleton, and so to lessen the bone conductivity, although, as a matter of fact, devices such as these afford but a trifling protection against the all-pervading thunder of a big gun.

It may be laid down, indeed, as a general rule that people who are sensitive to noise should plug the ears when exposed to it.

CONCUSSION DEAFNESS

This name is often applied to the deafness which follows exposure to one single loud sound, such as shell explosion, but it would be more proper to reserve it for those cases which follow a blow upon the head.

In warfare, a rifle or shrapnel bullet striking the skull, if not fatal, is frequently followed by nerve-deafness affecting chiefly the ear corresponding to the side upon which the wound has been inflicted. In civil life it is met with after head injuries, especially such as have caused concussion of the brain.

Symptoms.—Deafness varying in degree up to absolute loss of hearing is noticed as soon as the injury is received, or as soon as the patient recovers consciousness, and it is frequently accompanied by a persistent high-pitched tinnitus. In the few cases I have tested, the vestibular reactions have been also impaired.

In degree, the loss of hearing varies from a moderate falling-off to absolute deafness, affecting one or both ears.

Prognosis.—The prognosis of true concussion deafness is not good. It tends to persist.

Treatment.—No treatment is of any avail to restore the hearing.

Deafness from Fracture of the Base of the Skull.—When, as is often the case, the line of fracture traverses the petrous, it generally passes through the cochlea, and leads to absolute deafness on the affected side, frequently associated with facial paralysis.

If the membrane is torn, blood and, it may be, cerebro-spinal fluid flow from the external meatus, in which case steps should be taken to render the meatus as clean as possible, and to render the ear aseptic.

The deafness is permanent, complete and irremediable.

SYPHILITIC DEAFNESS

During the time of activity of the secondary syphilitic throa lesions the middle ear may become infected with pyogeni organisms and a purulent otitis media develop, with the usua history of such an infection.

But in addition to the septic infection, it would seem that there may also at this stage be a syphilitic infection of the middle ear which manifests a tendency to induce inflammatory and porotic changes of the bone of the labyrinth capsule, resembling microscopically those of otosclerosis. The cochlea is involved secondarily. These changes have been described as occurring in congenital syphilis (J. S. Fraser).

Otherwise, the pathological process underlying the familiar deafness of acquired syphilis does not seem to be very clearly known. The general impression is that the spirochæte induces low inflammatory changes in the cochlea, and that the acoustic nerve may be the seat of neuritis, or become involved in syphilitic meningitis of the base.

The deafness of congenital syphilis generally appears some time between puberty and middle age, but it may set in in childhood, and indeed it is admitted that syphilis is one of the common causes of the early deafness that leads to deaf-mutism. (See p. 587.)

In the acquired disease it is most usual as a late secondary phenomenon, but its appearance may be delayed until long after all other syphilitic manifestations have disappeared, and in such cases the true cause of the deafness is very difficult to discover.

Symptoms.—The cochlear and vestibular systems seem to be simultaneously affected, as the tinnitus and deafness are combined with attacks of vertigo sometimes very severe. Thus, the Ménière syndrome may at times be an expression of syphilis.

Tinnitus is prominent; it appears early, and it is loud and persistent.

The deafness, which usually affects one ear more than the other, is of the type of nerve deafness, but the presence of middle ear changes not infrequently introduces the obstructive element, and then the tests reveal a mixed deafness.

The vestibular reactions show impairment, or give an almost completely negative response.

Progress is rapid, and in the severer cases, tragically so, as the hearing in both ears may be entirely obliterated in a year or two, or even in less time. But the disease frequently stops short of complete destruction, and improvement, according to Politzer, may follow suitable treatment. Even complete deafness, he states, may be recovered from. The writer's experience is much less fortunate.

Diagnosis.—Severe, bilateral, progressive, nerve-deafness in a patient showing other signs of syphilis is to be diagnosed as almost certainly syphilitic. But it is more difficult to be convinced when the ear changes occur in a patient otherwise healthy. The Wassermann test may aid us in coming to a decision.

Children suffering from nerve deafness, even if slight, should always have the Wassermann reaction taken, as syphilis is the most common, though not, of course, the only cause of nerve deafness in early life.

Treatment.—It is generally agreed that the onset of syphilitic deafness is a signal for the vigorous prosecution of anti-syphilitic remedies, especially by mercury and iodide of potassium.

The writer has seen absolute deafness follow within three months of the exhibition of salvarsan in a case of moderate syphilitic deafness, and his experience is not unique. Deafness and vertigo therefore may be regarded as contra-indicating the salvarsan group of drugs.

Politzer recommends hypodermic injections of 2 per cent. sol. pilocarpin for eight to fourteen days, and then if no benefit follows, anti-syphilitic treatment should be commenced.

Urban Pritchard for congenital syphilitic deafness has found great benefit from blistering the mastoid processes.

Deafness from Epidemic Cerebro-Spinal Meningitis.—In this disease the infection travels from the lepto-meningeal spaces by the sheath of the auditory nerve to the labyrinth, where a destructive labyrinthitis, which may be purulent, is set up. When such cases recover, the hearing is found to be completely and permanently destroyed, and as this disease is not infrequent in infancy and childhood, we have in epidemic meningitis one of the common causes of deaf-mutism.

Diagnosis.—To elicit a history of meningitis sometimes requires considerable skill in cross-questioning, but if the story given is that of unconsciousness lasting for several days, followed by absolute deafness, the diagnosis is probably that of epidemic meningitis.

Treatment.—No treatment is of any avail.

Deafness from Mumps is, fortunately, a very rare complication of this common disease.

It is sudden in onset, and often, but not always, appears without any vestibular disturbance, and without tinnitus. Only one ear is affected as a rule, but the deafness is absolute. The sudden and complete destruction is striking. The patient may go to bed well, and wake up in the morning with one ear absolutely deaf.

The vestibular responses in the few cases I have seen were normal.

Deafness following orchitis with a high temperature has been recorded by the writer as being also probably due to the virus of mumps, as orchitis may occur without parotid involvement. And the same may be true of deafness following ovaritis.

The pathology of mumps deafness is unknown ; its prognosis is unfavourable ; and there is no method of treating it.

Deafness in Typhoid Fever.—The deafness which occurs in the course of typhoid fever is due to the general toxic influence of the illness upon the neurones. It nearly always disappears as the patient recovers.

Deafness from Myxœdema.—In myxœdema, both the conducting apparatus and the receptive apparatus are affected by the disease, and as a consequence, the deafness is of the "mixed" type.

Unless on the look-out for it, one is apt to miss myxœdema, but the heavy countenance with its fixed flush and the loss of hair in eyebrows and scalp, ought to be sufficient to attract attention, and to lead to diagnosis.

The effect of thyroid feeding on the deafness is good.

Progressive Labyrinth Deafness.—(Primary atrophy of the Acoustic Nerve of the Cochlea.)—Under this name is described a variety of progressive nerve deafness which affects males chiefly, of over middle life. It is referred to atrophy of the acoustic nerve fibrils in the cochlea and nerve-root. The vestibular nerve-endings have been unaffected in the cases examined microscopically.

Arterio-sclerosis, syphilis, chronic nephritis, and also middle ear inflammations have been blamed with inducing it, but it may possibly be related to early senility.

It tends to progress to absolute deafness, but it may become arrested.

Senile Deafness.—(Presbyacusis).—All forms of deafness become more pronounced with advancing years, but senile changes in the auditory perceptive mechanism are normal, and induce a nerve deafness which may become very severe in old people.

The tests reveal a very considerable depreciation of the hearing for high tones as well as the other signs of nerve deafness.

It is probable that, as with other phenomena of senility, senile deafness may at times set in while the patient is otherwise still comparatively youthful.

Senile deafness has an insidious onset, and tinnitus is seldom or never experienced.

Deafness from Drugs.—The auditory nerve is peculiarly susceptible to the action of certain drugs. Quinine is the best known of these, to cause tinnitus and deafness, which if the remedy has been given in large doses for a prolonged period may even become permanent. Indeed, it is often difficult to determine whether the deafness of a malarial patient may be due to the disease or to the remedy. The salicylate group also induces tinnitus and deafness, but I have never known permanent damage to follow this drug. Chronic alcoholic patients often become deaf, doubtless from alcohol, and according to Wingrave, the excessive use of tobacco may have the same effect.

Lead, mercury, and carbon disulphide have likewise a deleterious influence upon the hearing, and we have already mentioned the action of arsenic and the salvarsan group of drugs upon the auditory nerve (p. 71).

Hysterical Deafness is not a common form of hysteria, and in the writer's experience, excluding for the moment the deafness of shell-shock, it has most frequently been found in patients who already were the subjects of deafness, the real malady obviously suggesting the hysterical aggravation. But hysterical deafness certainly does sometimes occur in people whose hearing has been quite normal.

The deafness has frequently a sudden onset, and may follow a shock. It shows one or two characteristics which serve in most cases to render its nature quite evident. Thus, the deafness for conversation is worst when the patient first comes into the aurist's presence; as the interview proceeds, it tends to get much less noticeable.

The tests, again, often reveal decided anomalies. The tuning-fork, for example, may show the bone-conduction to be greatly diminished, and yet the hearing for high-pitched sounds, as estimated by the monochord or by Galton's whistle, may be perfectly normal.

Finally, according to the writer's results, the caloric reaction is normal, and that test frequently has all the effect of a "shock" treatment, inasmuch as it may restore the hearing!

Other stigmata of hysteria in the patient at the moment or in the history should also be sought for. But these may be absent, and the first hint of the nature of the deafness may be its sudden disappearance.

The treatment is that of hysteria. Sometimes, as in other hysterical paralyses, the symptom may prove to be very obstinate.

SHELL-SHOCK DEAFNESS

The Great War has made us acquainted with cases of absolute deafness in men who have been exposed to the devastating experience of high-explosives. Of the real character of the lesion in such cases, excluding such obvious damage as that of ruptured membranes, dislocated ossicles, and canalicular and cochlear hæmorrhages, we have no definite information, and even the question whether these cases are to be regarded as organic or hysterical or functional is not quite settled.

A number of authorities are inclined to look upon the vestibular reactions as affording a means of distinguishing organic and incurable shell-shock deafness from the functional or hysterical and curable type, and although this attitude has evoked violent protests from some quarters, many observers continue to believe in the test as a useful practical guide, and base their treatment upon it.

If the vestibular reactions in a case of absolute deafness following shell-shock are negative, that is if the responses are minimal, * the assumption that the deafness is organic and likely to persist is, on the whole, probably correct. Cases have been recorded certainly where some hearing was regained in these conditions, but the amount of recovery and the rarity of such cases scarcely invalidate the above statement, according to the writer's experience and belief.

The reason is that an explosion sufficiently powerful to destroy the end-organ for hearing, by intra-cochlear hæmorrhage, e.g., is also likely to have affected the vestibular end-organ as well, since, as we have already seen (p. 579), probably both are, under certain circumstances, exposed to the same stimulus through the endolymph.

If, on the other hand, the vestibular reactions are normal or approximately so, it is obviously impossible to determine *absolutely and from that fact alone* that the deafness is purely functional or hysterical. We have repeatedly seen, for example, that the vestibular reactions may be preserved in organic deafness

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* Complete abolition of all vestibular response to caloric, galvanic, or rotatory tests is rare under any circumstances.

(e.g. in mumps deafness, see p. 582), and although the toxic agent of mumps with its selective action upon the auditory nerve or its end-organ is a very different agent of destruction from the massive indiscriminating air-push of a high explosive shell, still it is conceivable that the canalicular system on the one part, or the cochlea on the other, may occasionally escape disruption although its adjoining neighbour suffers it.

Nevertheless, although a normal vestibular reaction does not infallibly certify the deafness of every case in which it is found to be functional in nature, it may, in the writer's opinion, be regarded as strongly supporting that diagnosis.

Although, therefore, we are not justified in branding as "hysterical," still less as "malingerer," any soldier with absolute deafness following shell-shock in whom the vestibular responses are normal, we are, nevertheless, encouraged to persevere with such remedies as moral suasion and hypnotism, because there is a strong chance that the patient will recover.

Shock-treatment, as by faradism, is ill-borne by these patients, just as we found in aphonia from shell-shock. And indeed, as a matter of fact, even the caloric and rotatory tests are liable to upset the patient's nervous system for hours after, and should be but cautiously employed.

There is another feature which favours the view of the functional character of such cases, and that is the general demeanour and bearing of these men. Their nervousness and intentional coarse tremors bear a close resemblance to the phenomena of "Railway spine"—familiar to insurance and railway medical officers.

Simulated Deafness—Malingering.—When a patient has anything to gain from being deaf, the possibility of deafness being simulated should always be considered.

In such cases one of the first signs to arouse the otologist's suspicion is the detection of an acute though covert watchfulness on the part of the patient. In responding to tests by the tuning fork, for example, he will reply in the negative a little too soon—before, for example, the base of the tuning fork is actually in contact with the mastoid in testing the bone conduction.

When the deafness is said to be unilateral and absolute, one may deceive the patient by means of two long tubes, one in each ear and united, like the rubber tubes of a binaural stethoscope, near the observer's mouth. The tubes are led behind the patient and by obstructing one or other tube as he speaks, the observer can test either ear as he wishes, unknown to the patient.

Bárány's Noise Machine Test.—When the noise-machine (Fig. 141) is held in the ear, and the person speaks aloud, he naturally adopts a loud tone, so as to drown the noise of the machine. This fact is utilized in the detection of simulated deafness. The subject is made to read aloud from a book, and while he is doing so, the noise-machine is started at his deaf ear. If he is really quite deaf, he will proceed with his reading in the same tone after as before the noise began. If, however, the deafness is simulated, he will instinctively raise his voice when the noise begins.

Neurasthenic Deafness is rare, although in neurasthenic states, a blowing tinnitus synchronous with the pulse is common. So also is vertigo, sometimes severe, but generally mild and evanescent. The genuine labyrinth storm with sickness and deafness may develop. The true nature of these phenomena is evinced by their transitory character and by their association with the other characteristics of the condition.

Neurasthenic deafness, when it does occur, is characterized by a rapid tiring of the auditory sense, so that at the beginning of the examination the hearing may be only slightly impaired, but as the examiner proceeds with his tests, the deafness will be found to get worse. Contrast this with hysterical deafness.

DEAF-MUTISM

If the power of hearing is congenitally absent, or if it is lost in early life, the child will be a deaf-mute. In the latter case, mutism follows if the deafness sets in before the age of from four to seven years, according to the intelligence of the child and the care exercised by its parents and teachers.

Etiology.—In the *congenital* type the influence of heredity is very powerful, and the defect is a family characteristic. Consanguinity has been blamed for the condition, and no doubt it will accentuate any tendency to deaf-mutism.

The pathological findings in the ear of congenital deaf-mutes embrace almost every possible defect in the hearing apparatus from auricle to brain, and which of the many defects it is that is hereditary is at present unknown.

In the *acquired* variety, the etiology and pathology are both clearer, since the causes chiefly blameable are the ordinary destructive diseases of the middle ear and labyrinth ; that is to say, suppuration of the middle ear extending to the labyrinth ;

epidemic cerebro-spinal meningitis; and congenital syphilis. A large percentage of the cases owe their deafness to middle ear suppuration following scarlet fever, diphtheria, and the other exanthemata in early infancy. Owing to this threat, acute suppuration of the middle ear in infants is a grave disease.

Symptoms.—Curiously enough the hearing of the congenital variety is not so utterly abolished as in the acquired group. Quite often it is possible by testing with forks of different pitches, by means of bells, and by whistles, to discover some modicum —some "islet of hearing in the sea of silence." And it is possible, sometimes, to educate this modicum, or at least to turn it to good purpose in the education of the patient.

The reason is that we are dealing in this group with the still remaining effects of a long-past destruction, or with a failure to develop simply. The condition is negative.

In the acquired group, on the other hand, the disease which induced the deafness is active and its effects are progressive. The condition, therefore, is positive. And as a result the process continues to the point of complete abolition of function.

The vestibular tests also reflect the same features. The responses are absent in 30 per cent. only of congenital deaf-mutes, while in the acquired cases they are absent in 70 per cent. (Alexander and Kreidl).

Diagnosis.—In deaf-mute families the defect is looked for and quickly discovered by anxious parents, but the acquired type may be overlooked unless the parents are very observant, until the child's slowness in speaking attracts their attention.

At early ages, the hearing is best tested by ringing bells, whistling, and such noises as do not stimulate the acute tactile sensation of these patients.

The ordinary deaf-mute is a person of acute intelligence, and in diagnosis, must be discriminated from those forms of idiocy which are associated with dumbness.

In the absence of family records, it is often difficult to distinguish between a congenital and an acquired case. But the evidence of the mother, if she is intelligent, as to whether or not the child ever seemed to be able to hear, may be sufficient to clear up the point.

Prognosis.—As has already been remarked, the prognosis in the congenital form is less hopeless than in the acquired.

Treatment.—*Preventive.*—Families in which deaf-mutism is hereditary should not inter-marry. For this reason the partial or entire segregation of deaf-mutes, or their separation from the general community is strongly to be deprecated. Every effort must be made to bring active disease to a standstill, and to treat the deafness. Cases are on record where seemingly hopeless deaf-mutes have regained a considerable amount of hearing by such means.

The treatment of deaf-mutism, nowadays, is by means of lip-reading to train the child to take his place in society, able to speak and able to understand what the normal individual is saying. This training is now wisely undertaken by the Education Authorities of this country, and the age at which the special education is begun is compulsorily fixed at not later than seven years. As Macleod Yearsley has pointed out, however, the necessities of the case demand that the education should be begun by the special teacher as early as the age of three years. The treatment is not restricted to absolute deaf-mutes; children who are too deaf to benefit by the class teaching in the ordinary schools are also sent to the special schools. (See below.)

THE TREATMENT OF THE SEVERER GRADES OF DEAFNESS

Deafness, as a defect, may be roughly divided up into seven grades :—

I. *Slight Deafness.*—Hearing lost for faint sounds; in conversation, asides and confidential half-hints are lost or are half-heard and half-constructed.
II. *Mild Deafness.*—Hearing lost for low conversation, for the *pianissimo* passages of instrumental music; for subdued tones in church or theatre.
III. *Moderate Deafness.*—Hearing lost for conversation not addressed directly to patient; for all distant speech in public; for household noises of moderate intensity.
IV *Severe Deafness.*—Hearing for ordinary conversational tones lost; raised voice necessary.
V. Hears only shout close to the ear.
VI. Absolute, or *nearly Absolute Deafness* to the voice, although some bone-conduction may still be present, and hearing for thunder and similar loud sounds.
VII. *Absolute Deafness* to all sound.

The last final stage is rare as an adult defect. It is generally syphilitic in origin.

It is noteworthy that in the higher grades of deafness there is generally nerve-deafness present to a greater or lesser degree.

Artificial Aids may be tolerated to help conversation in Grade III. In Grades IV. and V. they are necessary. In Grades VI. and VII. they are of no use.

Deafness in Schoolchildren.—As a practical guide in determining whether the hearing of a child is sufficient to permit of his being educated at an ordinary school, Kerr Love lays down the following *six-foot rule:* If a child does not hear a whispered speech with at least one ear at a distance of six feet, he cannot profitably remain in the ordinary classes of an elementary school, and should be sent to special classes or schools for the deaf.

Artificial Aids.—Undoubtedly the most popular aids are the pocket combinations of telephone and megaphone of which there are a large variety in the market. The patient should choose his own pattern, and should give it some days' trial before acquiring it. Many people object to the loud, adventitious noises heard, and to the intensifying of the unimportant sounds of everyday life. For this it is possible to vary the instrument in certain directions.

The old-fashioned conversation-tubes and horns have still many votaries, and instrument-makers have excelled in the art of the clever concealment of such instruments.

As to the effect of artificial aids upon the hearing, it is difficult to express an opinion. Probably they are beneficial, keeping alive a faculty which, if we may argue by analogy with vision, would otherwise tend to atrophy and complete loss.

Re-education of Hearing.—With this name there has recently been instituted a mode of treating the higher grades of deafness by means of musical sounds graded in certain ways. The method, unfortunately, has fallen into the hands of unqualified practitioners, before we have had sufficient opportunity of arriving at a definite opinion upon it.

As far as the writer's experience goes, the method has had an extremely restricted success. After the course of treatment is finished, patients with otosclerosis and other severe deafness often realize that their hearing has brightened up. They can hear sounds better for a few days. Then it slips back to its former level again.

A few cases of quite remarkable improvement have been reported. But they have almost certainly been largely functional, the dormant hearing sense being roused by the treatment. The writer believes that this dormant element bulks largely in many cases of grave deafness.

Ear Disease and Life Insurance.—There are many diseases of the ear which threaten life, and any one of these should lead to rejection of the patient for insurance. Such are acute suppuration of the middle ear, and chronic suppuration with signs suspicious of extension to labyrinth or brain.

A case of simple chronic discharge without granulations or polypi; and free from pain or other threatening symptoms, especially when the discharge is slight in quantity, and there is no sign of cholesteatoma, might be accepted at a higher rate; and the same is true of patients who have undergone the radical mastoid, if, say, a couple of years has elapsed since operation without serious signs.

Active cholesteatoma should lead to complete rejection.

Cases with acute suppuration may be deferred until well, and the same applies to simple acute mastoiditis, and the other acute complications.

Such grave diseases as tubercle or malignant disease, of course, would lead to rejection.

Absolute deafness should be considered a bar, and should lead to an increased premium, as such people are more liable to meet with accidents on account of their deficient hearing. This applies more to acquired than to congenital deafness, as in the latter the patient is much more alert with his eyes.

THE PREVENTION OF DEAFNESS

As we have frequently had cause to remark, the treatment of ear disease in its established states is relatively seldom so successful as to remove the deafness which that disease has induced.

Consequently, of recent years more attention has been paid to the prevention of the ear-diseases which cause deafness. For this new orientation of the efforts of the otologist we are in this country particularly indebted to the activities of Macleod Yearsley and Kerr Love.

In this direction, the following matters should be carefully attended to:—

1. Adenoids and tonsils should be removed as soon as they begin to cause symptoms.

(Nearly all the middle ear diseases of later life, both suppurative and non-suppurative, commence during the earlier years of the school period.—Kerr Love.)

2. If obstructive deafness is not speedily cured by this operation, inflation should be methodically practised.

3. Acute suppuration of the middle ear should be properly treated, especially when it occurs in infants and young children, and when it follows the exanthemata. For this reason an aural surgeon ought to be attached to all Fever Hospitals.

Acute suppuration of the middle ear should not be allowed to become chronic.

4. The efficient preventive treatment of such endemic diseases as cerebro-spinal meningitis and syphilis will be followed by a reduction in the number of deaf children.

5. In adult life, nasal and naso-pharyngeal diseases should be treated with the view of preventing deafness. But it is necessary to remind the reader that unless there are signs of "catarrh," i.e., local infection of a low type, or of recurrent acute catarrhs, operation on throat and nose is not likely to prevent deafness, or to alleviate it if it is already present.

6. Families in which there is hereditary deaf-mutism or otosclerosis (stapedio-vestibular ankylosis) should not intermarry.

CHAPTER XV

THE NERVE COMPLICATIONS OF EAR DISEASE

In addition to the nerves proper to the cochlear, and to the vestibular end-organs, *the facial trunk* and several of its branches enter into close and sometimes immediate relationship with the ear, and are exposed to damage in ear disease. Further, the situation of the Gasserian ganglion of *the fifth nerve* in a shallow recess on the anterior surface of the petrous pyramid not far from the tip exposes that structure to the influences of suppurative bone disease, and finally *the sixth cranial nerve* (to the external rectus of the eye) traverses a dural compartment known as the "space of Dorello" medial to the tip of the petrous, where it is exposed to the effects of suppuration in the petrous bone.

OTOGENIC FACIAL PARALYSIS

is commoner than is generally supposed. Indeed, there are strong grounds for the belief that most cases of the so-called "rheumatic" facial paralysis are due to middle ear disease or to herpes zoster oticus.

The course of the facial nerve exposes it to infective neuritis in inflammations of the middle ear, labyrinth, and mastoid cells, as well as to injury in fracture of the base of the skull or in the mastoid operations.

It enters the internal auditory meatus along with and above the cochlear and vestibular divisions of the auditory nerve, leaving them at the fundus of the meatus, and traverses the petrous in a short, narrow tunnel close to the second spiral of the cochlea, to emerge at the hiatus Fallopii. Here is situated the geniculate ganglion, and at this spot the nerve trunk bends sharply backwards and outwards, entering the wall of the tympanic cavity, and passing on between the external semicircular canal above and the oval window below to the pyramid where it again bends, this time passing vertically downwards to emerge from the stylo-mastoid foramen into the parotid region, where it takes a forward course to reach the face.

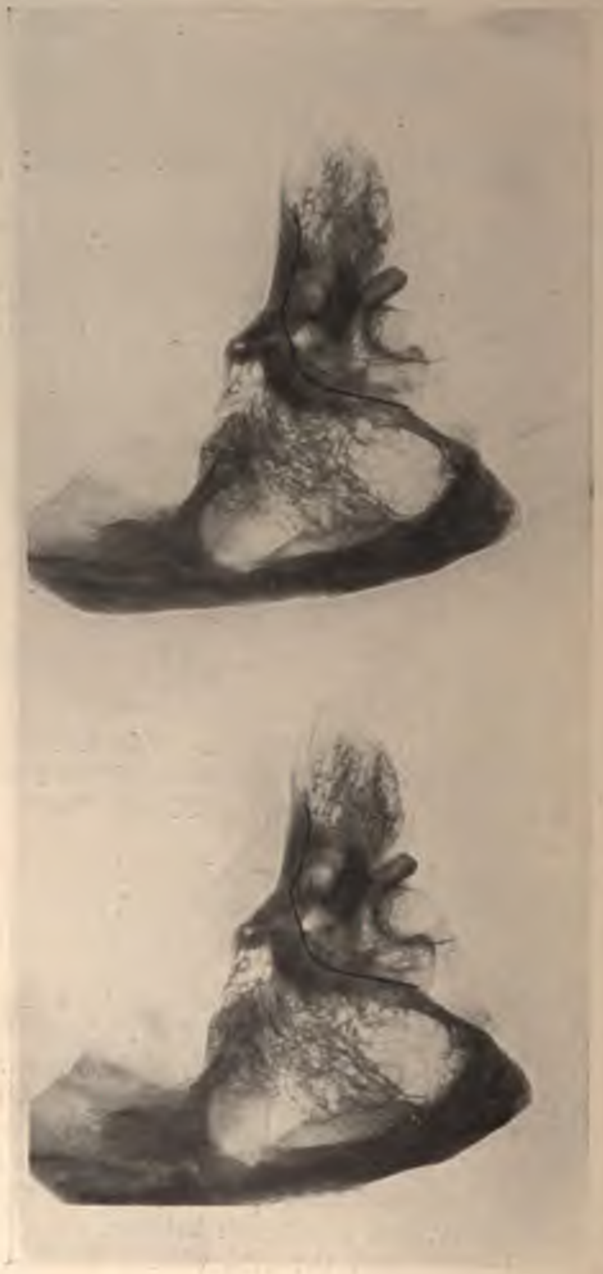

FIG. 196.—Stereoscopic skiagram (by Dr. Robt. Knox) of temporal bone of adult; from before backwards. The mastoid is pneumatic. The facial canal contains a wire. The mastoid antrum; the superior semicircular canal; and the whorls of the cochlea are all distinguishable.

In the petrous section it is exposed to injury in fracture of the base of the skull, and in necrosis of the labyrinth. At the geniculate ganglion it is paralysed in herpes zoster oticus. (See p. 442.) But it is in the wall of the middle ear that the trunk is most exposed. The Fallopian canal is here normally covered with a protective sheath of bone, but in a large number of cases the bony covering is deficient or absent, and then the nerve trunk is only separated from the tympanic mucosa by a thin membrane.

Thus, facial paresis or paralysis may result from any inflammatory disease of the middle ear, whether catarrhal or purulent. That being so, in all cases of facial paralysis the ear should be examined. In many, the ear symptoms are slight and evanescent, and the effect of the catarrh is most obvious in the paralysis.

Facial paralysis has not been reported as occurring in chronic middle ear catarrh or in otosclerosis.

In suppuration, it may attend either the acute or the chronic forms of the disease, sometimes varying in degree, but generally tending to complete paralysis. Its occurrence is an indication for immediate mastoid operation.

In its vertical descent to the stylo-mastoid foramen, the nerve lies close behind the tympanic membrane, and may be exposed here and paralysed by cerumino-cholesteatomatous plugs in desquamative external otitis, and in this segment also it is exposed to infection from adjacent mastoid and other cells.

Symptoms.—Facial paralysis in its fully developed form is unmistakable. Facial paresis, however, may be overlooked in its mildest forms, as when the weakness is limited to the orbicularis palpebrarum only, and is of very brief duration.

Symptoms of paralysis of the chorda tympani, loss of sensation on the anterior two-thirds of the corresponding side of the tongue, are sometimes experienced in acute suppuration, and after incision of the membrane, or the radical mastoid operation. But they are less frequently complained of than might be expected.

The *Prognosis* of facial paralysis depends upon the cause.

In fracture of the skull and in labyrinth necrosis, it is nearly always permanent. As a consequence of middle ear catarrh, it usually is recovered from in four to six weeks, if the catarrh is cured. In suppuration, the paralysis almost invariably persists until the mastoid operation is performed. If it persists after the operation, it is likely to be permanent.

The prognosis of facial paralysis from operative trauma can be roughly estimated from its time of onset. If the paralysis is observed for the first time at or immediately after the operation, probably the nerve trunk is severed and the paralysis permanent. But even in the worst cases, a certain amount of recovery, sufficient to conceal the deformity, usually takes place in one or two years.

If the onset is delayed for several hours, and is gradual in appearance, it will last from three to six months, and will probably get slowly well, although with some slight permanent weakness of the affected side of the face.

If the onset is first noticed a few days after operation, the cause is a neuritis of the intact trunk ; it will get well in four or six weeks ; and cases which do not pass beyond the stage of paresis, will recover completely in two or three weeks.

Treatment.—After removing the cause when that is possible, the nutrition of the affected facial muscles may be maintained by the faradic or galvanic current according as they respond to either. The eye should be protected against the results of exposure by a pad and bandage during the night.

Implication of the Gasserian ganglion (Fig. 189) or of some of its branches in suppurative disease of the petrous bone seems to be rare, but it is by no means unknown. The symptoms are neuralgic pains referred to the face and teeth, for which it is not unusual for patients to have sound teeth removed. The cases seen by the writer have occurred in acute suppuration of the middle ear with evidences of deep extension of the infection.

The *Treatment* consists in opening up the middle ear by a radical mastoid operation coupled with an extension of the operation where necessary.

Paralysis of the External Rectus (Abducens Paralysis) in suppuration of the ear is variously referred to local meningitis affecting the sixth nerve in its long course at the base of the brain, or to neuritis set up by the petrous suppuration as the nerve traverses the space of Dorello. Probably the latter is the correct explanation, as cells communicating with the antro-tympanic spaces may be found extending as far as the tip of the petrous.

Most of the cases get well after the mastoid operation is performed and efficient drainage instituted, but occasionally the paralysis is permanent.

CHAPTER XVI

AFFECTIONS OF THE MOUTH

THE LIPS

Hare-lip is a congenital fissure of the upper lip: *incomplete* if the tissues of the lip alone are involved; *complete* if the cleft extends into the nostril; *simple* if it affects the soft parts only; *alveolar* if the bony alveolus is also involved, and *complicated* if associated with the cleft-palate. When unilateral, it generally occurs on the left side; when double or bilateral, it is usually alveolar, and associated with complete cleft-palate (q.v.). In the latter case it is usual to find the isolated portions of the lip and alveolus attached to the end of the nose.

Herpes Labialis occurs in clusters of small vesicles on the cutaneous surface of the lip in the course of catarrhs, pneumonia, malaria, and other pyrexial states. The eruption is accompanied by heat and burning of the skin. Herpes may also appear on the inner aspect of the lip, or on the palate, fauces, or pharynx, where it takes the form of small, oval, shallow ulcers.

Eczema of the lip, acute or chronic, is, as a rule, secondary to irritating nasal discharges. The skin in this affection is red, swollen, infiltrated, and weeping. The morbid secretion, if allowed to dry, forms the familiar whitish crusts or scabs.

Impetigo Contagiosa appears in more or less circular and circumscribed spots, exuding a secretion, which, on drying, gives rise to the typical honey-coloured crusts. Ample evidence of its contagious nature can be obtained from an examination of the rest of the face, the scalp, and the hands.

Lupus of the lips occurs in the warty, ulcerating, and non-ulcerating varieties.

Lupus Erythematodes is less common on the lips than on the skin of the nose.

Syphilis.—The lip may be the seat of lesions due to primary, secondary, tertiary, and hereditary syphilis.

Primary Chancre is not uncommon. It occurs as a smooth, indolent ulcer, exuding a scanty sero-purulent secretion, and resting upon a mass of infiltrated tissue, which may involve the whole lip. The infiltration is less dense but more extensive than that accompanying hard chancre of the genitalia. The submental and submaxillary glands are generally enlarged.

Chancre is liable to be mistaken for **epithelioma.** The differences are as follows: Epithelioma generally affects the lower lip, chancre the upper; the surface of the epithelioma, if it has had time to ulcerate, is warty and irregular—the surface of chancre is smooth and flattened; in chancre the skin is more involved, in epithelioma the mucous membrane; finally, a short duration, the appearance of a sore throat and skin eruption, and an early enlargement of the lymphatic glands, are unmistakable signs of a syphilitic infection. The age of the patient may also be of some value in balancing probabilities.

Secondary Syphilis affects the lips in the form of mucous tubercles on the inner surfaces, and at the angles of the mouth. The secondary roseola of syphilis is rarely seen on the face.

AFFECTIONS OF THE BUCCAL CAVITY

Stomatitis.—Many varieties are described in the text-books; for the most part they are easy of diagnosis.

1. **Simple Catarrhal Stomatitis.**—*Causes:* Sepsis associated with carious teeth. Improper feeding and depressed general health predispose to the disease.

The mucous membrane of the gums becomes red, inflamed, tender, and invested with a delicate, greyish, filmy membrane, easily removed without damaging the underlying mucous surface. There are also present salivation, some pain on eating, and general *malaise.* Children are more subject to the complaint than adults.

2. **Ulcerative Stomatitis** has been described as an acute variety of the last named, but it is really quite a distinct disease. It frequently occurs in epidemic form amongst children in a family or in an institution. There are present some constitutional disturbance, soreness in the mouth, and salivation. On examination, small circular or oval ulcers are seen on the gums, which are swollen, livid, and bleed easily; on the lips; and not infrequently on the dorsum of the tongue. Each ulcer is surrounded by an areola of erythema, and the surface is covered with firm, yellowish exudation. The tongue is furred and somewhat swollen. The submental and submaxillary glands are

enlarged and tender. Spontaneous recovery is the rule, in a week or ten days.

Treatment.—Both these forms give way to simple mouth-washes such as, sol. hydrogen. peroxid. (10 vols. per cent.) tc. myrrh with potass. chlor.

The individual ulcers of the ulcerative variety may require to be touched with silver nitrate. In doing so, it is important to dry the ulcer and the mucous membrane around before making the application, in order to keep the silver salt from spreading.

3. **Aphthous Stomatitis (Thrush)** is characterized by the presence of small, whitish spots, composed of the mycelium and spores of fungus, the *Oïdium albicans.*

4. **Gangrenous Stomatitis (Cancrum Oris. Noma)** is a disease of childhood, occasionally following measles, scarlet fever, or diphtheria. It consists in the formation of a gangrenous patch on the cheeks or gums, surrounded by an intensely inflamed area of mucous membrane. The gangrene tends to spread with great rapidity, and from the profound toxæmia it induces is very frequently fatal. A similar condition occurs at times about the vulva.

The *treatment* of noma consists in the prompt application of fuming nitric acid to the edges of the gangrenous area.

5. **Mercurial Stomatitis.**—The signs of mercurial stomatitis are those of a severe stomatitis, with the addition of a more profuse salivation. If allowed to continue unchecked, deep ulceration of the gums with loosening and falling out of the teeth result, accompanied by grave general disturbances—tremors, emaciation, and asthenia.

The **Gums** participate, of course, in diseases affecting the mouth.

Alveolar Abscess is the formation under the periosteum covering the alveolar process of an abscess which originates at the root of a carious tooth. The tooth having died, the pulp cavity becomes septic, and an abscess forms at the apex of one of the roots. Accumulating here, and causing more or less pain, the pus burrows to the outer or inner side of the alveolar process, either by passing towards the neck of the tooth, under the periosteum, and so over the edge of the socket to the exterior of the bone, or else by making an opening from the apex of the socket directly through the bone. In the upper jaw the pus, burrowing, may penetrate the antrum of Highmore, and induce empyema.

In the lower jaw the pus may track outwards to the skin, and burst externally, leaving a depressed scar adherent to the ramus of the jaw. It may also burrow further afield, giving rise to deep cervical cellulitis behind and below the angle of the jaw, or it may pass in towards the root of the tongue to point in the region of the lingual tonsil. A large alveolar abscess may induce necrosis of the mandible.

Chronic abscess forms in connection with the dead tooth, the pus discharging through a minute sinus, marked by a small granulation in the outer or inner surface of the alveolar process. Fluctuating swellings on the hard palate are usually chronic alveolar abscesses.

The diagnosis of alveolar abscess presents, as a rule, no difficulty. The pain and swelling, the association with a carious tooth, and the presence of fluctuation, distinguish it from all other tumours. At times, however, particularly in the case of the long-fanged teeth of the upper jaw, swelling in the cheek precedes by some days the appearance of tumour and fluctuation in the mouth, and the diagnosis may then be a matter of doubt.

The *treatment* consists in opening the abscess, and in having the dead tooth from which it has originated removed.

Pyorrhœa Alveolaris consists in an extension of bacterial infection, with the formation of pus, from the margin of the gum and the tooth into the socket, between the bone and the root of the tooth. Little pockets are thus formed between the tooth and the bone, and these by extension and multiplication lead to atrophy of the gum (" recession ") and to absorption of the osseous alveolar process. The dental roots are thus exposed, and ultimately, if the disease progresses unchecked, the teeth loosen and drop out. The disease occurs quite independently of dental caries.

The diagnosis is in most cases easily made. The atrophied gums, loose teeth, often with exposed roots, and the pockets out of which pus can be pressed, form an unmistakable combination. The disease does not usually affect all the teeth unless it has existed for many years.

The importance of the disease from the oto-laryngologist's point of view lies in the fact that when the mouth is affected with pyorrhœa, it is in a virulently septic state, and that operations on the pharynx, larynx, and nose should not be undertaken until the pyorrhœa has been cured or rendered as nearly innocuous as possible.

The *Treatment* of pyorrhœa is a task for the dentist. The disease is extremely obstinate, and when advanced, can only be cured by removal of the affected teeth.

Dental Cyst occurs, it is said, as a sequel of dental caries, but this is doubtful, as it frequently forms in connection with an unerupted though sound tooth. It is most common in connection with the first upper molars and bicuspids. A small cystic bud attached to the root of a tooth to begin with, it slowly and painlessly enlarges, distending the bone of the alveolus, and thinning this osseous covering out until it becomes soft and fibrous, crackling along the edges on pressure.

The cyst may attain to a large size, growing at the expense of the antrum, and expanding the whole upper jaw if permitted to grow unchecked. It is filled with fluid, and is lined with a thick leathery membrane, which must be entirely removed if radical cure is to be obtained.

Diagnosis.—Do not mistake dental cyst for malignant disease of the antrum, or for suppuration of the antrum.

Treatment.—When small, the cyst is exposed in the mouth by incision through the gum over the sac; the thinned out bone is peeled off it as completely as possible, and the cyst is shelled out entire. The cavity left, fills up with granulation tissue, and cicatrizes over. Any unerupted, carious, or dead teeth should be removed.

When it is so large as to occupy the cavity of the antrum, the best treatment is to operate as for the radical antrum operation, and to remove not only the fibrous cyst wall and its lining but also the whole of its expanded bony covering, opening into the cavity proper of the antrum, and completing the operation by forming an antrostomy opening into the nose, and suturing the wound in the mouth.

If the cyst has grown so large as to cause facial deformity by expansion of the body of the maxilla, it should be eradicated as described in the last paragraph, but the facial deformity will persist.

Odontomes are tumours arising from the various dental tissues—enamel, follicle, papilla, etc. They are rare.

Epulis.—Any tumour growing from the alveolus may be termed "*epulis.*" As a rule, however, the name is applied, firstly, to fibroma arising from the periosteum—*simple epulis*; and secondly, to myeloid sarcoma arising from the medulla of the alveolar process—*malignant epulis.*

Simple Epulis is a red, smooth, fleshy, rounded or lobulated growth covered with mucous membrane, and containing frequently some bony spicules. It is most common in the lower jaw, its main mass resting on the external surface of the alveolar process.

Malignant Epulis occurs as a soft, rapidly-growing tumour, purple in colour, and rapidly ulcerating or fungating.

The *Treatment* of both is by removal. Myeloid sarcoma is not very malignant.

Necrosis of the jaw-bone may be caused by subperiosteal alveolar abscess secondary to dental caries, traumatism (with dentist's forceps, etc.), phosphorus-poisoning, tubercle, or may appear as a sequel to one of the infectious fevers.

The symptoms are those of abscess-formation, with persistent sinuses after the abscess is opened. Probing will reveal dead bone.

DISEASES OF THE TONGUE

Congenital Abnormalities.—Tongue-tie consists in an abnormal shortness of the frenum. It is a favourite bugbear of mothers and nurses, but only deserves attention when it interferes with suckling or speech.

Macroglossia, though sometimes acquired, is usually congenital. The hypertrophy is said to be due to lymph-angiectasis. The tongue is enlarged in all directions, protruding from the mouth, and indented by the teeth ; it is purple in colour, and becomes dry from exposure.

Wounds in the tongue are usually caused by the teeth. In this case the wound is transverse to the long axis of the organ. When produced by some sharp, foreign body, the wound is usually longitudinal.

Acute Parenchymatous Glossitis is a rare disease. It may be caused by mercurial poisoning, or by wounds in the tongue which have become septic.

Symptoms.—Pain on moving the tongue and swallowing is noticed, then the tongue rapidly becomes swollen. In a day or two it may become so large that the patient cannot withdraw it into the mouth. There is more or less fever and constitutional disturbance. Recovery after about a week's illness is the rule, but the inflammation may go on to form an abscess, or end in gangrene. These terminations are, however, exceedingly rare.

Ludwig's Angina is a form of septic cellulitis or erysipelas of the submaxillary region and neck secondary to a streptococcus or pneumococcus glossitis and pharyngitis. It is characterized by the formation of a hard, brawny swelling of the parts under the jaw and in the upper cervical region. The floor of the mouth, the fauces, the pharynx, and the larynx participate in the general phlegmonous swelling, and severe constitutional disturbances co-exist. There is grave danger to life from septicæmia, from œdema of the glottis, and later from exhaustion. Multiple abscesses form in the neck if the patient survives the early stage. (See also p. 41.)

Ulceration of the Tongue.—Ulcers of the tongue are not uncommon. They arise—(1) from the irritation of carious teeth, in which case they occur at the sides of the organ—the *dental ulcer;* (2) from dyspepsia, the so-called *dyspeptic ulcer*, which is found on the dorsum and is often very painful; (3) from *tubercular* disease, which is always secondary to phthisis; (4) in the tongue when affected with *leukoplakia;* and (5) in *epithelioma.*

Ulceration of the frenum is not uncommon in whooping-cough, from the fraying of the tense fold against the lower incisor teeth during the paroxysms of the cough.

Chronic Superficial Glossitis is usually the result of syphilis, but it certainly occurs in cases where no history of syphilis can be obtained. To account for the disease in these circumstances, we may suppose that it has arisen in consequence of some long-continued irritation, such as is produced by excessive tobacco-smoking, dental caries, dyspepsia, spirit-drinking, etc. The condition is of importance from the liability it manifests to end in epithelioma. Several varieties or stages in the disease are described, any or all of which may be found in the same patient:

1. The stage of localized hyperæmia, like the "smoker's patch."
2. The dorsum becomes invested with opaque, bluish-white, shiny patches—leukoplakia—generally surrounded by
3. Smooth, glazed, red areas, the "glazed red tongue" often occurring with
4. Cracks or fissures, and at times superficial ulceration or warty projections, the whole tending to end in
5. Epithelioma.

In this complaint the parenchyma of the tongue is affected as well as the surface, and as it tends to become sclerotic, the substance of the organ feels hard and thin when pinched between finger and thumb. The condition causes much discomfort from the hypersensitiveness of the surface to hot or spicy foods and drinks.

Treatment.—Locally, the regular use of sol. hydrogen. peroxid. (10 vols. per cent.) is very beneficial, but in the syphilitic variety of the disease most benefit accrues from the salvarsan remedies.

Syphilis of the Tongue.—The primary sore is rarely met with. Secondary manifestations occur on the tongue as mucous tubercles, fissures and ulcers, generally at the sides and tip, and in this stage a broad wart-like condyloma on the dorsum may be found.

Gummata of the tongue are fairly common. They occur as firm, fleshy swellings in the substance of the organ, towards the middle or posterior part, and near the middle line. If left untreated, they soften and break down, ulcerating through the surface, and forming roundish excavated ulcers, with a yellowish, sloughy, "wash-leather" base. There is neither induration nor excavation of the edges of the ulcer, however, and the movements of the tongue are quite free—features which, with the painlessness and the rapid progress of the disease, serve to distinguish the gummatous ulcer from epithelioma.

For *Treatment*, see p. 71.

Cancer of the tongue occurs in the form of squamous epithelioma. The disease most commonly starts as an ulcer at the edge of the tongue, towards the junction of the middle and posterior thirds, and frequently in association with a jagged tooth. But, as we have already noted, it not infrequently begins in the form of a red warty outgrowth from, or in a fissure of a tongue affected with chronic superficial glossitis. Cancer may also originate in the floor of the mouth, or in the tonsil, pillars of the fauces, etc., and attack the tongue secondarily by extension.

Early diagnosis is naturally of the utmost importance. Attention should be paid to the following points :—The epitheliomatous area is hard, and feels to the examining finger to be in or close under the surface of the organ. Such a condition in a tongue already the seat of leukoplakia should at once raise the question of cancer. Ulceration occurs early. The surface of the ulcer is sloughy, the edges undermined, notably indurated, and raised. Salivation and restricted movement of the tongue are symptoms which appear later, and become more and more marked as the disease advances. The pain is almost always very severe. The lymphatic glands liable to infection are the submental, the submaxillary, and the deep carotid nodes. Difficulty in mastication, speaking, and swallowing, mark the later stages of the disease.

In all cases of doubt, a portion of the suspected growth should be removed and examined microscopically. The test of treatment by iodide of potassium should never be altogether relied upon, since if there is syphilitic disease co-existing with the epithelioma, the improvement due to the disappearance of the former element may lead to the raising of false hopes and the loss of valuable time.

Treatment.—Operable cases are those in which the growth is limited to the substance of the tongue, and in which the glands, if enlarged, are discrete and non-adherent.

Operation consists in the removal of the tongue, together with the glands, whether enlarged or not, and the gland-bearing fascia of the submaxillary and upper cervical regions.

THE BASE OF THE TONGUE

is conventionally regarded as lying within the boundaries of otolaryngology. For the examination of this region, which lies beyond the foramen cæcum and the circumvallate papillæ, the laryngeal mirror is required. (See p. 80.)

The Lingual Tonsil consists of a number of rows of gentle elevations or folds of lymphoid tissue arranged antero-posteriorly on the surface of the tongue beyond the foramen cæcum and the circumvallate papillæ. The masses, which vary considerably in bulk in different individuals, are situated on either side of the middle line.

Hypertrophy of the Lingual Tonsil is commoner in adults than in children.

When *symptoms* are present they consist of reflex phenomena set up by irritation of the superior laryngeal nerve, the afferent nerve of this sensitive area, and include a feeling of fullness or of a foreign body in the throat; irritable cough; and, as a rarity, spasm of the glottis.

Whether or not reflex symptoms are set up from an enlarged lingual tonsil is one of the dead controversies of laryngology. There is no doubt whatever that such symptoms do occur, and that they are readily amenable to treatment.

Treatment.—Simple astringent gargles, and the painting of the base of the tongue at intervals of a few days with

℞	Ferr. Perchlor.	gr. 240.
	Glycerin. ad	℥i.—M

are often sufficient to subdue the irritation (Mark Hovell).

If these fail, the lingual tonsil may be cauterized with the galvano-cautery, a red heat being preferable to a white heat, in order to avoid reactionary hæmorrhage ; or diathermy may be employed.

Large Varicose Veins ramifying over the base of the tongue and lingual tonsil do not occur, but varicose dilatations of small venules are common, and devoid of any significance save when they give rise to slight hæmorrhage, which is liable to be mistaken for true hæmoptysis. In a typical case, the bleeding occurs when the patient stoops, or when he is sucking sweets, etc., and the usual story is that the blood " comes into the mouth " without coughing, vomiting, or retching. But in a case of hæmoptysis it is wise not to look upon dilated veins at the base of the tongue as the most likely source of the hæmorrhage until all other possibilities have been excluded.

Lingual Tonsillitis and Abscesses are considered at p. 34.

The base of the tongue is a favourite site for *gummatous deposit* and *ulceration,* which manifests the usual characters. (See p. 73.)

Lingual Thyroid, see p. 177.

Epithelioma is also common in this region, beginning generally at the juncture of the anterior pillar of the fauces with the tongue.

It tends to spread both towards the tongue and towards the tonsillar region, and its features, in general, are those of cancer of the pharynx. (See p. 62.)

Lymphosarcoma of the base of the tongue may also be mistaken for an enlarged lingual tonsil or for an accessory thyroid. In the case of sarcoma, the tumour is rapidly growing, and presents a nodular surface. A portion removed and microscopically examined will clear up any doubts as to its nature.

Treatment of Cancer of the Base of the Tongue.—The exposure of this region for the removal of growths at the base of the tongue may be effected by subhyoid pharyngotomy. (See p. 145.)

The excision of the growth may frequently be accomplished with the diathermy knife. (See p. 65.)

DISEASES OF THE FLOOR OF THE MOUTH

The under surface of the tongue and the floor of the mouth present for examination the frenum linguæ, with the openings of the submaxillary ducts (Wharton's) on either side of its attachment to the floor of the mouth. The sublingual salivary gland is marked by a round oval protuberance in each side of the floor on a ridge on the surface of which its many ducts open.

Sublingual Abscess is usually the result of wound infection. A turgid swelling forms in the floor of the mouth, presenting the usual signs of phlegmonous inflammation. The tongue may become swollen in sympathy, and inflammatory œdema of the larynx may occur.

Treatment.—The abscess is opened under local anæsthesia, by an incision through the mucous membrane, followed by a pair of forceps plunged into the swelling, opened, and withdrawn open.

All **cysts** in this situation are commonly and impartially termed "*ranulæ*," just as a hard tumour of the alveolar process of the jaw bone is called "epulis."

A ranula may form in connection with any of the salivary or mucous glands which open into the floor of the mouth. The most common ranula occurs in connection with the sublingual gland, the site of which, on one or both sides, is occupied by a tense bluish cyst, over which veins are seen to be coursing. As a rule, these cysts are about the size of a pigeon's egg, but they may be so large as to interfere seriously with the movements of the tongue.

When a ranula forms in connection with the submaxillary gland, the cyst can be plainly felt externally, just under the horizontal ramus of the mandible.

A mucous cyst in the floor of the mouth may simulate a sublingual cyst very closely, and some observers have been inclined to look upon this form of ranula as much more frequent than that caused by cystic dilatation of the salivary ducts.

Treatment.—The cyst should, if possible, be dissected out entire, as only thus can one be sure of its non-recurrence. But when large and thin-walled, the best practice is to remove a large part of its wall, picking it up in the floor of the mouth with forceps, and cutting a piece out with scissors curved on the flat. The little operation may be performed under local anæsthesia.

A **Dermoid Cyst** in the floor of the mouth is distinguished from ranula by its yellowish or orange colour, and by pitting of the wall of the sac on pressure.

Improper treatment of these cysts may result in the formation of a **dermoid fistula** in the neck.

Treatment.—The cyst should be dissected out as a whole. A general anæsthetic is required.

Among the other neoplasms found on the floor of the mouth are endothelioma, lipoma, papilloma, epithelioma.

Neuroses of the Tongue (Motor).—Tremors.—The tongue, being almost wholly composed of muscular tissue, provides us with an excellent clinical guide to general muscular tonus. In exophthalmic goitre, chronic alcoholism, and other diseases in which muscular tremor is a feature, the tongue exhibits the symptom as a coarse or fine fibrillar twitching, or as a trembling of the organ as a whole.

Paresis and Paralysis.—Unilateral paresis or paralysis may result either from a central or peripheral cause.

In paralysis affecting the organ as a whole, the cause is almost invariably bulbar paralysis. In this case, when well marked, the patient is unable to protrude the tongue, to chew his food properly, or to pronounce words distinctly. The condition should be carefully differentiated from immobility of the tongue arising from the presence of cicatricial, cancerous, or other adhesions binding it down to the floor of the mouth.

Paralysis of the hypoglossal nerve of one side is tested by asking the patient to insert his tongue into and against the cheek while we test the vigour of the act with a hand on the outside. The test is homo-lateral; that is to say, that when the pushing out of the left cheek is feebly performed, it is the left hypoglossal that is to blame, and *vice versâ.*

Anæsthesia of the anterior two-thirds of the tongue is due to paresis or paralysis of the lingual nerve, and may result from operative injury to the nerve trunk in the floor of the mouth [operation for salivary calculus, ranula, guillotine tonsillectomy, etc.].

Deficiency of taste, but not of simple sensation in this region is experienced when the chorda tympani nerve is severed as in paracentesis tympani, and other middle ear operations, and also along with facial paralysis in lesions of the facial trunk proximal to the geniculate ganglion.

Paræthesia of the tongue is a source of great mental distress when it appears, as the patient almost always believes the " pricking," " swelling," " numbness," or " pain " to be due to a cancerous growth.

DISEASES OF SALIVARY GLANDS

Salivary Calculus occurs more frequently in the ducts of sublingual and submaxillary glands than in Stenson's duct, as the secretion of these glands is thicker and contains more mucus than that of the parotid. The stone is usually spindle-shaped, and about the length of a large rice-grain, but it may be spherical and attain to a large size. It is made up of the carbonate and phosphate of calcium.

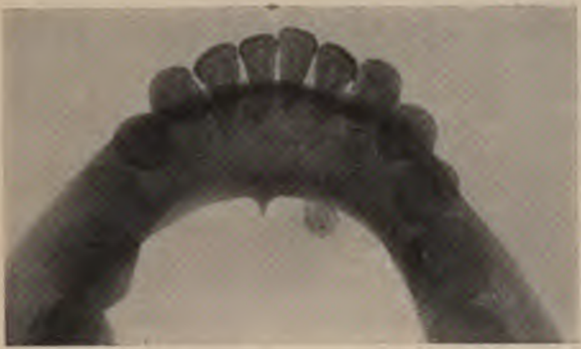

FIG. 197.—Skiagram (by Dr. Robert Knox) of Salivary Calculus (sublingual or submaxillary).—Case of Mr. G. J. Jenkins.

The *symptoms* produced are variable. Sometimes they clearly indicate the cause, as when there is a partial or complete obstruction of the duct with swelling of the gland at meals from distension. At other times, pain and thickening from chronic adenitis alone exist to direct attention to the gland.

Sometimes, under those conditions, the calculus can be felt on bi-digital examination, one finger inside the mouth, and the other externally on the submaxillary gland. But here also error is not unknown, as a gland enlarged from simple chronic adenitis may feel as hard as a stone.

If a fine probe can be passed along the duct, the calculus may be felt, but sounding in this way is rarely possible.

Occasionally, the presence of the stone leads to abscess-formation, and when the pus is discharged into the mouth, the calculus is voided along with it.

The X-rays, however, in expert hands may enable us to diagnose calculus with confidence. (See Fig. 197.)

Treatment.—The removal of the calculus may be more difficult than one expects. It is, therefore, advisable to operate under general anæsthesia.

Operation.—The mucous membrane of the floor of the mouth is picked up with dissecting forceps and an incision made into it of about an inch in length. If necessary, it may be deepened until the knife is heard to grate on the stone, but under the pressure of the knife the calculus may slip out of reach and be hard to find again. Aided by external pressure, the calculus is exposed sufficiently to allow of a curved, sharp spoon being inserted under it in order to scoop it out. Care should be taken to ensure the removal of all the calculous fragments.

The wound should not be sutured, but allowed to granulate, as sutures may lead to constriction of the duct and later on to stricture.

Inflammation.—While acute inflammation of the sublingual and submaxillary glands is only occasionally met with, **parotitis**, or inflammation of the parotid gland, is common.

Epidemic Parotitis (Mumps) is an acute, infectious disorder, affecting children for the most part, and occurring in epidemics. After an incubation lasting about three weeks, the disease manifests itself in painful swelling of one parotid, together with pyrexia and some constitutional disturbance. In a few days, the gland first affected gets well, and the other becomes swollen and tender. The diagnosis of mumps, which is not always easy, may be made from the extent of the situation of the swelling. The whole gland being involved, fullness and tenderness will be felt below and in front of the ear, and over the masseter muscle —the *pars socia parotidis*. The neighbouring lymphatics and the other salivary glands are likewise inflamed, and metastatic orchitis or ovaritis may supervene. Sudden absolute unilateral deafness is one of the rare sequelæ of the disease. (See p. 582.)

The *Treatment* of mumps is symptomatic. The patient is kept in bed while the fever lasts and the inflamed glands are kept warm.

The infection presumably travels from mouth to mouth by way of food utensils.

Simple Parotitis manifests itself in pain, swelling, and tenderness of one parotid gland. It is probably always due to septic infection spreading up from the mouth. It is predisposed to by a calculus in Stenson's duct, and I once saw it as the result of the invasion of the duct by a fine pillow-feather. The patient was an infant under the care of Dundas-Grant.

If the infection is of a virulent character, suppuration may occur, an event which may be suspected when the symptoms are acute, the fever high, and when the skin over the inflamed gland is dusky in colour and pits on pressure.

All inflammatory affections of the parotid, like those of the testis, are painful from the existence of a firm containing fascia, but when an abscess is forming, the suffering may be excruciating. Suppurative parotitis may occur in the course of pyæmia, or it may induce pyæmia by infecting the large venous channels which permeate the gland.

The pus from an abscess in the parotid tends to burrow deeply, and, following the lines of least resistance according to the disposition of the parotid fascia, may point in the pharynx, in the external auditory meatus, in the mouth internal to the ramus of the jaw, or it may pass down into the neck under the middle layer of the deep cervical fascia. Nevertheless, in spite of the strength of the external layer of the parotid fascia the pus not infrequently passes directly outwards through the skin over the gland.

If the abscess points and is opened over the parotid gland, the incision should pass through the skin only, the deeper parts being opened by blunt dissection, and the incision should be horizontal in order to lessen the risk of damage to the duct, as this accident almost certainly involves the production of the troublesome *parotid fistula*.

Chronic Adenitis of the Salivary Glands most commonly affects the submaxillary glands. It is most commonly induced by the presence of a calculus, but it is sometimes difficult to find an explanation for its presence unless, perhaps, it be assumed to be due to oral sepsis, as it is not infrequently found in association with pyorrhœa alveolaris.

Symptoms.—Pain, sometimes severe, is the chief symptom. The gland is swollen, and, generally speaking, soft, but occasionally it may be indurated to a scirrhous hardness. Its size and consistence are best made out on bi-digital examination.

Treatment.—Any possible cause, such as a calculus or pyorrhœa, should be removed. If no cause can be found, iodide of potass. in moderate doses may, albeit that the disease is non-syphilitic, restore the gland to its normal state, and cause the pain to disappear.

If this fails, and the symptoms continue, and are hard to endure, it may be necessary to remove the entire gland through an external incision. This operation is sometimes also undertaken when the gland has been disorganized through septic infection following a calculus.

Mikulicz's Disease is the name given to a condition in which all the salivary and sometimes also the lacrymal glands undergo chronic enlargement from lymphoid hypertrophy.

The condition is perhaps not a true disease entity seeing that it is known to occur in certain blood disorders, in combination with general enlargement of lymphatic glands, and of the spleen, as well as in syphilis, while a tuberculous variety of the salivary gland enlargement with tubercle bacilli in the tissue has also been observed.

Nevertheless, there are cases in which no other symptom or sign of disease is present except the chronic enlargement of the salivary and lymphatic glands, and to this group the name of Mikulicz's Disease is correctly applicable. It exists for years, and may occasion the patient much discomfort from the dryness of the mouth which it often induces.

NEOPLASMS OF THE SALIVARY GLANDS

Simple.—The tumour found most frequently in the parotid region is of a mixed character, containing fibromatous, myxomatous, and enchondromatous elements. Pathologists generally classify these tumours as endotheliomata. They are also met with in connection with the submaxillary gland.

In the parotid region, the growth occupies the position of the gland—that is to say, the main mass usually lies between the jaw and the sterno-mastoid, and supplementary growths intimately connected with the main mass are found to correspond with the *pars socia* on the cheek, while other extensions pass deep into the neck.

Typically, the tumour is of slow growth, is painless, and does not give rise to secondary involvement of the lymphatic glands. Interference with the movements of the jaw, if it occurs at all, is merely as a result of the bulk of the tumour. It should not be forgotten, however, that a simple parotid tumour may take on a malignant character.

Diagnosis.—Innocence may be diagnosed if the tumour is of slow growth, painless, and does not induce early facial paralysis in the case of the parotid.

Treatment.—The removal of the tumour by operation is called for, and in the submaxillary region is easily effected, the tumour shelling out in a mass. In the parotid, the result of the operation will depend upon the size and situation of the tumour. In any case, the dissection involves the risk of injury to the facial nerve which runs through the parotid gland from the stylo-mastoid foramen forward on a level with the lower border of the conchal cartilage of the ear to break up into the plexus of its branches on the cheek.

Malignant (Sarcoma or Carcinoma).—The usual signs of malignancy characterize these tumours—viz., rapid growth, invasion of neighbouring tissues, early adhesion of skin to the tumour, the appearance of facial paralysis, and the termination in ulceration and fungation, with extension of the disease to the lymphatics or to the internal organs.

Diagnosis.—It may be difficult to decide whether a parotid tumour should be regarded as simple or malignant. The most important signs of malignancy are the early involvement of the skin and the early appearance of facial paralysis.

The *Treatment* entails the entire removal of the malignant growth, and of the cervical glands and gland-bearing connective tissue.

AFFECTIONS OF THE PALATE

(See also p. 24.)

Cleft-Palate is a congenital defect of the roof of the mouth resulting from the non-union of one or both of the horizontal palatal processes with the ethmo-vomerine septum, which descends vertically from the under surface of the fronto-nasal process and the base of the skull.

If one palatal process fails to unite with the septum, a *unilateral cleft* is the result. The left side is most liable to this defect. When both fail to unite with the septum, a *median cleft* results, which may or may not be divided into two parts by the projection of the septum into the cleft.

As the union of those fœtal processes normally proceeds from before backwards, the different varieties of cleft-palate depend upon the extent to which this union occurs. Thus, the cleft may involve the uvula alone—*bifid uvula;* secondly, it may extend through the velum and through the hard palate as far as the anterior palatine canal; or, lastly, deviating to one or other side at the junction of the premaxillary with the maxillary bone, it may extend through the whole palate, in which case it is complicated with hare-lip of the same side. When the cleft is bilateral—i.e., when the septum fails to unite with either palatal process—double alveolar hare-lip co-exists, and the central portion of the intermaxillary bone—os incisivum—may be displaced, and attached to the tip of the nose.

A child suffering from cleft-palate cannot suck, and if fed with a spoon, returns the liquid through the nose. After speech is learned, the typical articulation of cleft-palate is heard, the explosive letters, dentals, labials, and gutturals being imperfectly produced. The exposure of the nasal mucous membrane results in chronic rhinitis, with crust formation and the loss of smell and taste.

After successful operation, these symptoms are considerably modified, and may, indeed, be removed. It is a general rule that the presence of cleft palate is a contra-indication of the adenoid operation. This applies even after the cleft palate has been successfully operated on.

Ulceration of the palate may be—

1. *Simple*, as in stomatitis ;
2. *Syphilitic :* (*a*) superficial (secondary) ; (*b*) deep, with caries-necrosis of the bone (tertiary).
3. *Lupoid*.
4. *Tuberculous*, usually secondary to tuberculous disease of the bone ; and
5. *Malignant*, generally epithelioma.

Stomatitis ulcers are discrete, circular, and multiple. They are easily cured by a touch of lunar caustic.

The **secondary syphilitic sore** appears along the free edge and margin of the soft palate, and forms part of the secondary pharyngitis. (See p. 70.)

The deep syphilitic ulceration is due to gummatous deposit, affecting either the hard or the soft palate. In the bony palate the gummatous deposit gives rise to a swelling in the palate always in the middle line. Unless it is just on the point of ulcerating, this swelling is smooth, painless, and firm. Left untreated, a typical gummatous ulcer rapidly forms, communicating with the carious area in the bone, through which a probe can be passed into the nose. This perforation goes on enlarging indefinitely, or until the existence of the sore leads the patient to seek advice. As a result of treatment, the ulcer heals, leaving circular perforations, large or small according to the stage at which treatment is begun.

It is seldom difficult to distinguish between the gumma of the hard palate and other forms of necrosis. Alveolar abscess from a carious tooth may induce necrosis, but in that case, the disease is unilateral, and implicates the alveolar process chiefly.

In the soft palate, the gumma is also usually situated in or near the middle line. Not infrequently the original deposit takes place on its superior surface, and perforation has occurred before the patient seeks advice.

In this region, perforation leads to nasal speech, and sometimes to liquids returning through the nose on swallowing.

Treatment.—Immediate constitutional anti-syphilitic treatment. (See p. 71.)

Tuberculosis of the hard palate is rare. Tuberculosis affecting the soft palate may be part of the disease in the pharynx. (See p. 75.)

Neoplasms of the Palate include endothelioma, adenoma, and fibroma, all of which are easy to remove.

Epithelioma is usually due to extension from the pharynx, but cases have been recorded in which a large area of the mucosa of hard and soft palate becomes the site of infiltration in which the occurrence of ulceration is delayed, and in which, as a consequence, the epitheliomatous nature of the infiltration has remained unsuspected. In the cases seen, the mucous surface was red, glazed, and thickened.

The removal of epithelioma involves the free removal of the soft palate (p. 391). If the disease has attacked the bone, the case must be considered inoperable.

CHAPTER XVII

THE LYMPHATIC VESSELS AND GLANDS OF THE HEAD AND NECK

There is probably no region in the body where the lymphatic glands are so frequently enlarged and diseased as in the head and neck; and there is certainly no region in the body where the sound surgical rule is so often forgotten: **When a lymphatic gland is enlarged, search for, find, and remove the focus of infection.** In the case of the nodes of the upper cervical chain which drain the pharyngeal and tonsillar regions, to carry this rule into effect is seldom a matter of difficulty, since the source of infection will generally be found in hypertrophied or fissured tonsils, or in carious teeth. But now and then we meet with cases of " enlarged glands " in the neck in which it is difficult, if not impossible, to hit upon the primary focus. In these cases a painstaking examination of the scalp, face, eyes, nose, naso-pharynx, mouth, pharynx, larynx, and ears, should be made and every possible source of infection removed. For, with few exceptions, enlargement of the lymphatic glands does not occur spontaneously. Perhaps even the disease known as lymphadenoma arises as a result of an infection from without, received through a breach of the surface. It is therefore important that we should be well acquainted with the various groups of lymphatic glands in the neck, and with the area each group drains.

The occipital glands, on either side, lie just below the occipital attachment of the occipito-frontalis muscle. They drain the occipital region of the scalp.

The mastoid glands, lying over the centre of the mastoid process, receive lymph from the parietal and posterior temporal portions of the scalp. An abscess of one of these nodes may be mistaken for the mastoid abscess of middle ear disease. It is distinguished by its superficial position, shorter history, and by the presence of the primary centre of infection (eczema, etc.) in the scalp.

The parotid glands are situated superficial to and within the substance of the parotid gland. The more superficial members of the group drain the anterior temporal and parietal regions, the cheek, eyelids and auricle. One of these, situated about a finger's breadth in front of the tragus, is often enlarged in diseases of the auricle. The deep parotid glands belong to the deep cervical chain. They receive lymph from the intra-

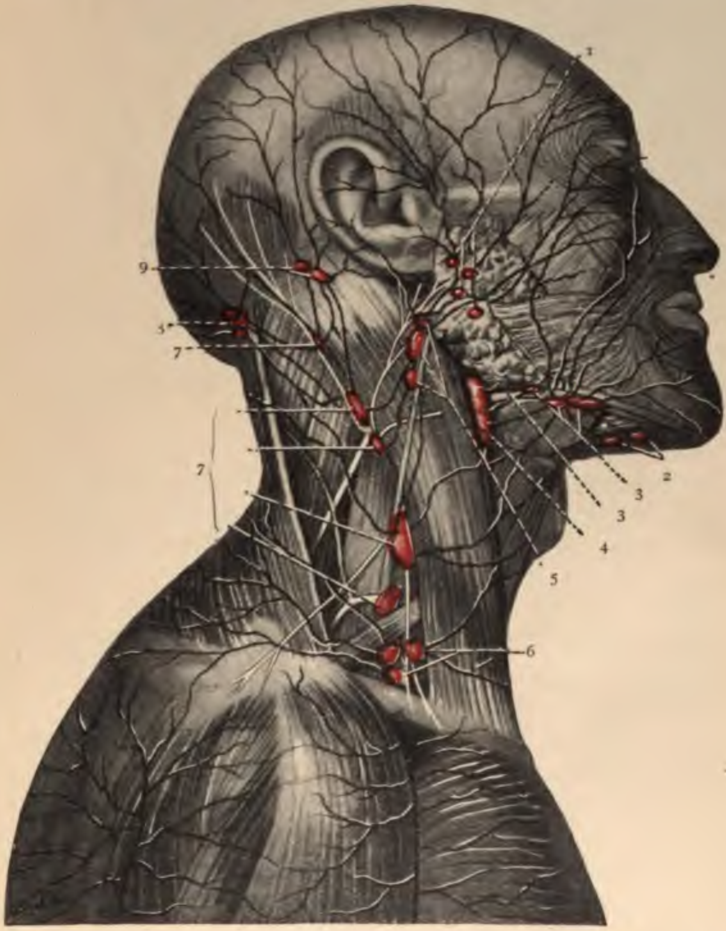

FIG. 198.—The Lymphatic Glands of the Head and Neck with the sterno-mastoid in position (from Cunningham's Textbook of Anatomy).

(1) Anterior auricular glands. (2) Submental. (3) Submaxillary. (4) An upper deep cervical gland. (5) Superficial cervical. (6) Supraclavicular. (7) Upper deep cervical (posterior group). (8) Occipital. (9) Posterior auricular or mastoid.

cranial region, and, by way of the **post-pharyngeal nodes** (see p. 44), they drain the pharynx and posterior parts of the nasal fossæ. They communicate in their turn with the lower members of the deep cervical chain.

Submaxillary Glands, within the sheath of the submaxillary salivary gland, may be felt, when enlarged, under the ramus of the jaw. They are supplied with lymph from the salivary glands and floor of the mouth, the lips, the anterior part of the tongue, and the cutaneous surfaces of the nose and frontal region. In dental caries affecting the lower molars they are generally enlarged.

The Submental Glands, one or two in number, lie superficial to the mylohyoid, and drain the lower lip, chin, and lower incisor teeth.

In the pharyngo-maxillary space, close to the pharynx, lie the deep **Internal Maxillary Group,** which drain the orbit, the palate, the greater part of the nasal cavities, the antrum and upper jaw, the deeper parts of the cheek, the back part of the tongue, and part of the pharynx.

All the foregoing groups, draining the important cavities of the head and face, pass their lymph on to two chains of glands lying along the external jugular vein and the carotid sheath respectively, and known as the superficial and deep cervical chains.

The Posterior Cervical Group extends downwards in the posterior triangle of the neck, just under the platysma, and so superficial to the deep cervical fascia. One or two members of the chain may be felt high up in the posterior triangle of the neck, just below the occipital region. This chain receives communications from the sub-occipital, the mastoid, and the submaxillary glands, and drains also the ear and the surface of the neck. Lower down in the neck it empties into the deep cervical chain, and in the supraclavicular region, communicates with the supraclavicular glands, and further outward with the maxillary glands.

The **Deep Cervical Chain** in contact with the carotid sheath directly or indirectly receives all the lymph of the head and neck, all the other groups ultimately pouring their lymph into this series. The most important members of the chain are: (1) Those lying behind and below the angle of the jaw, which, as we have seen, are almost invariably enlarged when the tonsils are hypertrophied (p. 50). The glands in this situation also enlarge secondarily to septic and inflammatory diseases of the pharynx, and to cancerous disease of the tonsils, palate, and tongue. (2) The other group of importance is that which becomes infected in cancer of the larynx. It lies on a level with the thyroid cartilage.

(In the lower neck both the superficial and the deep cervical

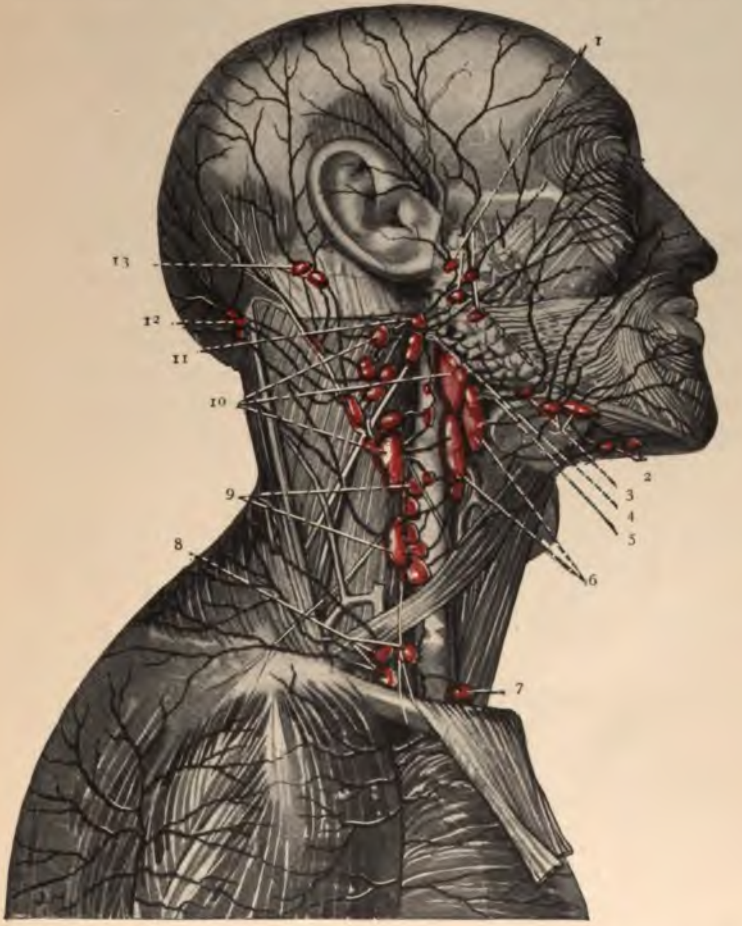

FIG. 199.—Lymphatic Glands of the Head and Neck after removal of the Sterno-mastoid (from Cunningham's Textbook of Anatomy).

(1) Anterior auricular glands. (2) Submental. (3) Submaxillary. (4) Cut end of external jugular vein. (5) Common facial vein. (6) Medial superior deep cervical—(glands of the angle of the jaw—tonsillar glands). (7) Medial inferior deep cervical. (8) Supraclavicular. (9 and 10) Deep cervical. (11) A superficial cervical gland. (12) Occipital. (13) Posterior auricular.

chains communicate with the supraclavicular lymph glands. These may become the seat of secondary deposits in mammary cancer by direct extension of the disease over the clavicle, and they are occasionally enlarged in gastric cancer as a result of the malignant growth from the abdomen, extending upwards in the thoracic duct. They sometimes become infected in cancer of the œsophagus.)

Two other lymph glands in the neck call for mention: one, a small gland lying on the trachea, under the isthmus of the thyroid gland, which enlarges in subglottic cancer of the larynx; and the other a single gland situated between the two layers of the deep cervical fascia in the suprasternal notch.

In the lymphatic systems of the head and neck, just as we know is the case elsewhere in the body, the intercommunication between the different sets of lymphatic vessels and glands is so free that infection may travel very far from its source, frequently passing glands which we should expect to be infected, and attacking others which, according to anatomical teaching, do not drain the diseased area at all. Thus, the glands in the superficial cervical chain are not infrequently enlarged and tender in catarrhal pharyngitis, and epithelioma on one side of the tongue may be productive of secondary involvement of the lymphatic glands on the opposite side of the neck.

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